A STUDY OF SEIZURE IN STROKE

Submitted in partial fulfilment of the requirements towards the conferment of

BRANCH – I D.M. NEUROLOGY

Of

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

DEPARTMENT OF NEUROLOGY

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI

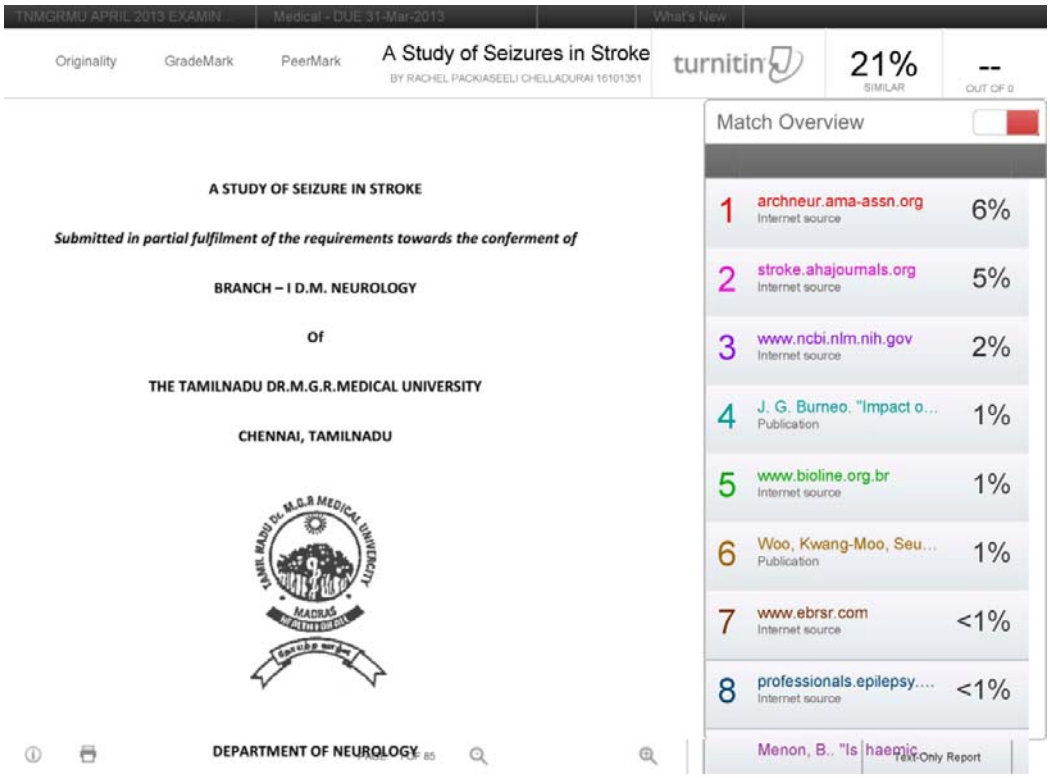
AUGUST 2013

CERTIFICATE

This is to certify that this dissertation entitled **"A study of Seizures in Stroke"** submitted by **Dr.C.Rachel Packiaseeli** appearing for D.M., Degree examination in August 2013 is a bonafide record of work done by her under my direct guidance and supervision in partial fulfilment of regulations of the Tamil Nadu Dr.M.G.R.Medical University, Chennai– 32. I forward this to the Tamil Nadu Dr.M.G.R.Medical University, Chennai.

Prof.Dr.S.Saravanan, M.D., D.M.,
Professor & HOD
Department of Neurology
Tirunelveli Medical College
Tirunelveli -11.

The Dean,
Tirunelveli Medical College,
Tirunelveli -11


TNMGRMU APRIL 2013 EXAMIN...
Medical - DUE 31-Mar-2013
What's New
Originality
GradeMark
PeerMark
A Study of Seizures in Stroke
BY RACHEL PACKIASEELI CHELLADURAI 16101351
turnitin
21%
SIMILAR
--
OUT OF 0
A STUDY OF SEIZURE IN STROKE
Submitted in partial fulfilment of the requirements towards the conferment of
BRANCH – I D.M. NEUROLOGY
Of
THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY
CHENNAI, TAMILNADU
DEPARTMENT OF NEUROLOGY
Match Overview
1 archneur.ama-assn.org Internet source 6%
2 stroke.ahajournals.org Internet source 5%
3 www.ncbi.nlm.nih.gov Internet source 2%
4 J. G. Burneo. "Impact o... Publication 1%
5 www.bioline.org.br Internet source 1%
6 Woo, Kwang-Moo, Seu... Publication 1%
7 www.ebrsr.com Internet source <1%
8 professionals.epilepsy.... Internet source <1%
Menon, B.. "Is haemic
Text-Only Report

TIRUNELVELI MEDICAL COLLEGE
TIRUNELVELI,
STATE OF TAMILNADU, INDIA
PIN CODE:627011
Tel: 91-462-2572733, 2572734 Fax: 91-462-2572944
Under the Directorate of Medical Education, Government of Tamilnadu.
Estd:1965

Institutional Ethical Committee

Certificate of Approval

This is to certify that the Institutional Ethical Committee of this College unanimously approves the Thesis /Dissertation/ Research Proposal submitted before this committee by Dr. **C.RACHEL PACKIASEELI, M.D,** *a* **DM POSTGRADUATE IN NEUROLOGY** *in the Department of* **NEUROLOGY**, *of Tirunelveli Medical College /Hospital, Tirunelveli titled "* **A STUDY OF SEIZURES IN STROKE"** *registered by the* IEC *as* **055/DM**/IEC/2011 *dated.* **25.02.2011**. *The Investigator is hereby advised to adhere to all the stipulated norms and conditions of this ethical committee.*

Issued on this
Date
25.02.2011
Under Seal

Secretary

Secretary,
Ethical Committee,
Tirunelveli Medical College,
Tirunelveli-11.

TIRUNELVELI MEDICAL COLLEGE
TAMILNADU
INDIA
TIRUNELVELI-11

DECLARATION

I **Dr.C.Rachel Packiaseeli** do solemnly affirm that this dissertation entitled **"A study of Seizures in Stroke"** is done by me at the Department of Neurology, Tirunelveli Medical College Hospital, Tirunelveli -11, during the period 2010- 2012 under the guidance and supervision of **Prof.Dr.S.Saravanan, M.D., D.M.,** Professor and Head, Department of Neurology, Tirunelveli Medical College.

This dissertation is submitted to the Tamilnadu Dr.M.G.R.Medical University towards the partial fulfilment of requirements for the award of D.M., degree in Neurology.

Place: Tirunelveli

Date:25.03.2013 **Dr.C.Rachel Packiaseeli**

ACKNOWLEDGEMENT

It gives me great pleasure to acknowledge all those who guided, encouraged and supported me in all the successful completion of my dissertation.

I whole heartedly thank **THE DEAN,** Tirunelveli Medical College for having permitted me to carry out, this study at the Department of Neurology, Tirunelveli Medical College Hospital, Tirunelveli-11.

First and foremost I wish to thank **Prof.Dr.S.Saravanan, M.D., D.M.,** (Neurology) Professor and Head of the Department, Department of Neurology for his constant guidance, motivation and valuable suggestions throughout the period of this work.

I owe my heartiest thanks to the **Associate Professor Dr.P.K.Murugan, M.D., D.M.** (Neurology), for his support and expert guidance.

I sincerely thank my **Assistant Professors Dr.V.Sriramakrishnan, M.D., D.M.** (Neurology), **Dr.M.Radha, M.D., D.M.** (Neurology), Department of Neurology, for their guidance and encouragement which enabled me to complete this study.

I would like to thank **Dr.A.Charles Pon Ruban,D.P.H.,** Tutor, Department of Community Medicine who helped me in statistical analysis of the study results.

Last but the most, I thank all my patients who participated in this study, for their cooperation which made this study possible.

CONTENTS

SL.NO	TITLE	PAGENO
1.	Introduction	1
2.	Aims of the study	2
3.	Materials and methods	3
4.	Methodology	6
5.	Review of Literature	13
6.	Observation, Analysis and Results	27
7.	Discussion	46
8.	Conclusion	65
9.	Summary	66
10.	Bibliography	67
11.	Appendix	

1. Proforma
2. Canadian neurological scale
3. Charlson Deyo Index
4. Master chart

INTRODUCTION

Seizures are a common phenomenon after stroke. Despite being first recognized more than a century ago, many questions regarding seizures in stroke remain unanswered. Stroke related seizures are a neglected topic and generally considered as a benign complication occurring in the course of a progressive and longstanding cerebro vascular disease. Differences in the study design, definition of late or early seizures, target population, inclusion and exclusion criteria, and data on imaging limit a direct comparison of the seizures and may explain the contradictory results in the literature.

The incidence of post stroke seizure in India is 13%.[9] There have been very few prospective studies in stroke related seizures from the Indian subcontinent. This study was designed to determine the time of onset, semiology, recurrence, impact of comorbid conditions, mortality and the anatomical location of stroke in relation to seizure.

A STUDY OF SEIZURES IN STROKE

AIM OF THE STUDY:

1. To study the semiology of seizures in stroke.
2. To analyze the occurrence of seizures in relation to stroke subtype
3. To study the time of onset of seizures in stroke.
4. To study the anatomical location of the lesion in stroke and the occurrence of seizure.
5. To analyze the recurrent seizures occurring in stroke.

MATERIALS AND METHODS

The present study was carried out at the Department of Neurology, Tirunelveli Medical College Hospital, Tirunelveli.

Study Design: This study is a single centre observational prospective hospital based study.

Period Of Study: September 2010 to February 2013

Ethical Approval: The study was approved by the Institutional Ethical Committee as 055/DM/IEC/2011 dated 25/02 /2011

Consent : An informed consent was obtained in those patients included for the study.

Sample Size: A total of 100 cases of seizures with stroke were included in this study.

Inclusion Criteria:

Patients aged above 16 years presenting with seizures associated with stroke were included in this study.

Exclusion Criteria:

1. Children and adolescents less than 16 years of age.
2. Patients with history of seizures prior to the occurrence of stroke
3. Stroke like presentation due to Neuro surgical causes like AV malformation, tumour, trauma and brain abscess.
4. Acute and chronic CNS infections manifesting as arteritis.
5. Patients with cortical venous thrombosis and venous stroke.
6. Stroke due to drug addiction and substance abuse.
7. Post-cardiac arrest resuscitation state.
8. Seizures associated with stroke as a sequlae of pregnancy related complications
9. Unwilling and non-cooperative patients.

Limitations of the study

1. The study was limited to patients presenting with seizures after stroke only.
2. The study included arterial strokes only.
3. The cases were followed up for a minimum period of 6 months with a mean period of follow up of 15 months. Hence the actual recurrence rate and response to long term AEDs could not be elicited.

4. Information bias

In aphasic patients history was elicited from the available informant and subsequently confirmed with a close care taker. Seizures are reported by the care takers were recorded. Some would not have noticed the onset of seizures whether focal or generalised.

Patient developing seizure in the hospital premises was seen immediately after a call from the concerned ward and the seizure was subsequently confirmed by interrogating with the resident in charge of the ward.

METHODOLOGY

After obtaining consent either from patients or relatives, all the patients in the study group were evaluated by complete medical history, neurological examination and routine baseline investigations. Axial CT brain was done in all patients, MRI was done in affordable patients.

Clinical history:

Clinical history was recorded from either the patient or his / her relatives. Name, age and sex of the patients were recorded. Special emphasis was given to the history regarding the seizure. The time of onset of seizure in relation to stroke, semiology of the seizures, whether the seizures were single or multiple, presence of recurrent seizures, details regarding the stroke - whether thrombotic or haemorrhagic or cardioembolic were recorded. Associated comorbid conditions like diabetes mellitus, systemic hypertension, pre existing cardiac illness, renal diseases, metabolic abnormalities, presence of systemic infections were taken into account. Inter-ictal EEG was done in 75 patients only due to technical difficulties.

Clinical examination:

Detailed neurological examination was done in all patients at the time of admission. Vitals were recorded. Neurological examination

included the level of consciousness, language impairment and the amount of motor deficit. Presence of metabolic disturbances like hyperglycaemia, hypoglycaemia, hyponatremia and hypocalcemia were recorded and correlated with the onset of seizures.

Seizures in stroke were defined as those either at the onset (or) after stroke in a patient without a prior history of seizure disorder. Seizures were classified[8] 'early', if the initial seizure (the first seizure after stroke) occurred within two weeks of stroke. Seizures were classified 'late' if the initial seizure occurred after two weeks of stroke. Early onset seizures were further classified as 'immediate' if seizures occurred within 24 hours of stroke onset. Recurrent seizures were defined as those occurring atleast two weeks after the onset of initial seizure. The seizures were considered multiple if the patient has 2 or more than two seizures.

Semiology:

Seizures were categorized as per the recommendations of the ILAE as generalized, simple partial, partial with secondary generalization or complex partial seizures. Presence or absence of status epilepticus was also noted.

Type of stroke:

The seizures were analysed in relation to the type of stroke - ischaemic or haemorrhagic. Of the ischaemic group, patients with a demonstrable cardiogenic source of emboli were noted and included in the embolic group.

Radiological assessment:

The following parameters were recorded from the CT brain

1. The type of stroke (ischaemic or haemorrhagic)
2. Side of the lesion
3. Location of the cortical infarcts – location of the lesion was classified[32] as follows.
    a. Frontal lesion – involvement of frontal, fronto parietal, fronto temporal, fronto parieto temporal
    b. Parietal lesion – includes parieto temporal, parieto occipital, and temporal
    c. Occipital lesion – includes occipital, occipito temporal and occipito parietal
4. The depth of the lesion was defined as cortical, subcortical or cortical and subcortical.

5. The size of the infarct was classified[9,32] as small <5cm, large >5cm, quantified from the greatest diameter on that CT slice showing the largest area involved.
6. The intra cererbral haemorrhage was classified[62] as
    a. deep ICH (basal ganglia, thalamus and internal capsule)
    b. deep ICH with intra ventricular haemorrhage
    c. deep ICH with lobar extension
    d. deep ICH with lobar extension and IVH
    e. lobar ICH (frontal, temporal, parietal occipital)

    The cortical involvement in intra cerebral haemorrhage was defined as any bleed that extended to cerebral cortex.

7. Volume of ICH

    The volume of ICH was measured according to ABC / 2 method[18] where A is the greatest haemorrhage diameter from CT, B is the diameter 90 degrees to A, and C is the approximate no.of the slices of CT multiplied by slice thickness.

    The ICH was classified[32] as

    - Small (0-29ml)
    - Large (30ml or more)

Electro Encephalo Gram:

The EEG findings were categorized[32] as follows

Type I – normal

Type II - presence of diffuse slowing

Type III – focal slowing with or without diffuse slowing

Type IV – focal spikes and sharp waves

Type V – Periodic lateralized epileptiform discharges (PLEDS)

The EEG findings were correlated with early Vs late onset seizures and with recurrent seizures.

Antiepileptic drugs:

Anti epileptic drugs were prescribed to all patients who were admitted during the study period with early onset seizures or with late onset seizures. Patients were prescribed either carbamazepine or phenytoin in appropriate doses. Injectable forms of benzodiazepine or phenytoin were used in patients admitted with multiple seizures and who were unconscious at the time of admission.

Assessment of stroke severity:

The Canadian Neurological Scale (CNS) (see appendix 1) was used for assessment of the severity of stroke. The CNS is a simple tool for use in the evaluation of the neurological status in the acute phase of stroke. The CNS evaluates 10 clinical domains including mentation (level of consciousness, speech and orientation) and motor function (face, arm and leg). Scores from each sections were summed to provide a total score out of the possible 11.5. Lower scores indicate more severity. The CNS scores can be reliably converted into NIHSS scores using the following validated conversion model (developed by Nilanon et al, 2010):

NIHSS Score = 23 – (2 x CNS score)

The CNS can be used prospectively and retrospectively (Goldstein et al).

In the present study, CNS was categorized into mild (CNS score >7), severe stroke (CNS score ≤ 7) Martin et al.

Comorbid conditions:

The presence of comorbid conditions associated with the seizures in patients with stroke were recorded. Past H/o CVA, presence of diabetes and its complications, systemic hypertension, presence of cardiac illness, inter current infections, renal diseases, metabolic disturbances etc

are among the important comorbid factors studied. Their association with seizures were correlated.

The Charlson – Deyo index[7](see appendix 2) was used to quantify the comorbidities. It is a summary score based on the absence or presence of seventeen medical conditions. It has been studied in several stroke related studies. A score of zero indicates no associated comorbidity and higher scores indicate a greater burden of comorbidity.

Mortality:

The number of deaths during the study period was studied. The probable risk factors for mortality was also studied.

REVIEW OF LITERATURE - SEIZURES IN STROKE

Stroke is one of the commonest cause of seizures in the elderly and seizures are among the most common neurological sequlae of stroke. Seizures can occur at the onset or may follow strokes.

Epidemiology

As early as 1864, Jackson recognized seizures as a complication that frequently occurred during the recovery phase of stroke. About 10% of all stroke patients experience seizures from the onset of stroke until several years later. In population studies, stroke is the commonly observed cause of epilepsy in adult population older than 35 years[17]

Timing of seizure in relation to stroke:

Approximately 10% of patients with stroke had seizures at some time after their stroke. In the Seizures After Stroke Study (SASS) , a prospective multicentre study conducted among university hospitals in Canada, Israel, Italy and Australia, 8.3% of stroke patients had seizures[18]. In this series, more than are half of seizures occurred on the first day of stroke. 80% of seizures occurred by the first month.

Among 1000 patients in the data bank collected in Girona, Spain, five percent of patients had seizures during the first 48 hours after CVA[31]

Gupta and colleagues analyzed the timing of seizures following stroke[32] . In their series of seventy patients with seizures after ischaemic stroke, one third of seizures occurred within the first two weeks, 90% had seizures with in the first twenty four hours. Nearly three fourths of seizures occurred within the first year of stroke. Only two percent had developed seizures more than two years after stroke. Patients who develop early seizures after stroke have higher in hospital mortality rate[40].

Seizures at the onset of stroke:

The occurrence of seizures at the onset of stroke has been well established[22]. The frequency had varied between 2 and 18% depending in the study and stroke type. In a large scale autopsy study, 13.8% of patients with intracerebral haemorrhage suffered seizures at the onset of stroke compared to 7% in the ischaemic stroke[23] . Superficial cortical lesion are more likely to precipitate seizures at the onset of stroke.

Seizures in relation to the type of stroke

Patients with intracerebral haemorrhages have seizures more often than with infarcts. In the Lausanne stroke registry, 7% of patients with intracerebral hemorrhage had seizures during acute stroke compared with

less than one percent of patients in the ischaemic strokes. Subcortical slit haemorrhages are most often associated in the seizures[24]

Among patients with ischaemic stroke those patients who had large and haemorrhagic infarcts have the most chance of developing seizures[(19,25)]

Patients with cardio embolic infarcts, have a much higher frequency seizures, than those with large artery occlusive infarcts. In supra tentorial infarcts, patients with cardioembolic stroke had a relative risk of 5.14 of developing early seizures than in patients non cardiac origin embolic stroke[26] . Earlier studies suggesting a relationship between cardiac embolism and seizures were observational and were performed before the availability modern imaging techniques.

In SASS(Stroke After Seizures Study), patients with cardiac embolic stroke were not at elevated risk of first seizure or recurrent seizure[27]. As per the data from Lausanne stroke registry[28], none of the 137 patients with probable embolism had seizures. Similarly data from the Stroke Data Bank Study[29] conducted by the National Institute of Neurological Disorders and Stroke (NINDS) showed that there was no association between seizures at onset and presence of a cardiac source of embolism.

In SASS, seizures were reported in 2.6% of patients with lacunar stroke. Results of functional neuro imaging and EEG showed that seizures in the background of lacunar stroke may be a reflection of concurrent cortical involvement[27]

Seizures in regard to stroke location

Patients with lesion at the cerebral cortex have a higher incidence of seizures than those with only subcortical lesions[19, 25] . Seizures may occur with subcortical involvement, a possible consequence of the release of glutamate from injured thalamocortical neurons. Lobar site is considered to be more epileptogenic in haemorrhagic stroke analogous to cortical involvement in ischaemic stroke. In a study by Faught et al, the incidence of seizure was highest with lobar bleeding into lobar cortical structures (54%), low with haemorrhage in the region of basal ganglia (9%) and absent with thalamic haemorrhage. Caudate involvement of basal ganglia and temporal or parietal involvement within cortex predicted seizures[30]

Recurrence of seizures in stroke

From the available data from stroke register about 5 – 20% of all individuals who have stroke will have subsequent seizures[27, 31] . Patients with early onset seizures were nearly eight times more likely to develop

late post ischaemic seizures and sixteen times more likely to develop epilepsy. A prospective study found seizure recurrence is 55% in late onset seizures[27] similar to that observed in other studies with longer follow up period[33,34] . Multivariant analysis has also proved that late onset (more than 2 weeks) seizures are an independent risk factor for epilepsy[27] . One retrospective study showed that 80% of patients with recurrent seizures were either not taking anti seizure medications or had sub therapeutic blood levels[32].

An underlying permanent lesion is responsible for higher frequency of epilepsy is patients with late onset than early onset seizures. Post stroke epilepsy develops in 35% of patients with early onset post stroke seizures and in 90% with late onset post stroke seizures. In case of haemorrhagic stroke, the risk of developing epilepsy is 29% with early onset seizures and 93% with late onset seizures[35].

Semiology of seizure in relation to stroke

Data regarding the subtype of seizure (simple partial, complex partial, partial with secondary generalization or generalised tonic clonic) in studies are limited by the retrospective design of most of the studies and are potentially confounded by interviewer and recall bias. Upto 63% of seizures may not be recognized by the patients[36] . Approximately 50 to 90% of early onset seizures are simple partial seizure[27,37,38,39]. In

contrast, one study reported higher frequency (50%) of GTCS without focal onset in early onset seizures[40] . Status epilepticus is a life threatening complication of stroke. Stroke accounts for 25% of cases of status epilepticus is few series[41] . A hospital based study found that 0.14% of patients with ischaemic or haemorrhagic strokes had developed status epilepticus.

Classification of post stroke seizures

Seizures are classified as early onset or late onset, according to the timing after the cerebrovascular accident. An arbitary cut off point of two weeks after the presenting stroke has been recognized to differentiate early onset and late onset post stroke seizures. There is no clear established pathophysiological basis exists for the two week cut off point. Early onset seizures are furthur classified as immediate - if the seizure has occurred within 24 hours of stroke. Recurrent seizures are defined as the seizures occurring at least 2 weeks after the onset of first seizure. Post stroke seizure is defined[15] as "single or multiple convulsive episodes after stroke and related to irreversible (or) reversible cerebral damage due to stroke regardless of time of onset of seizure following stroke".

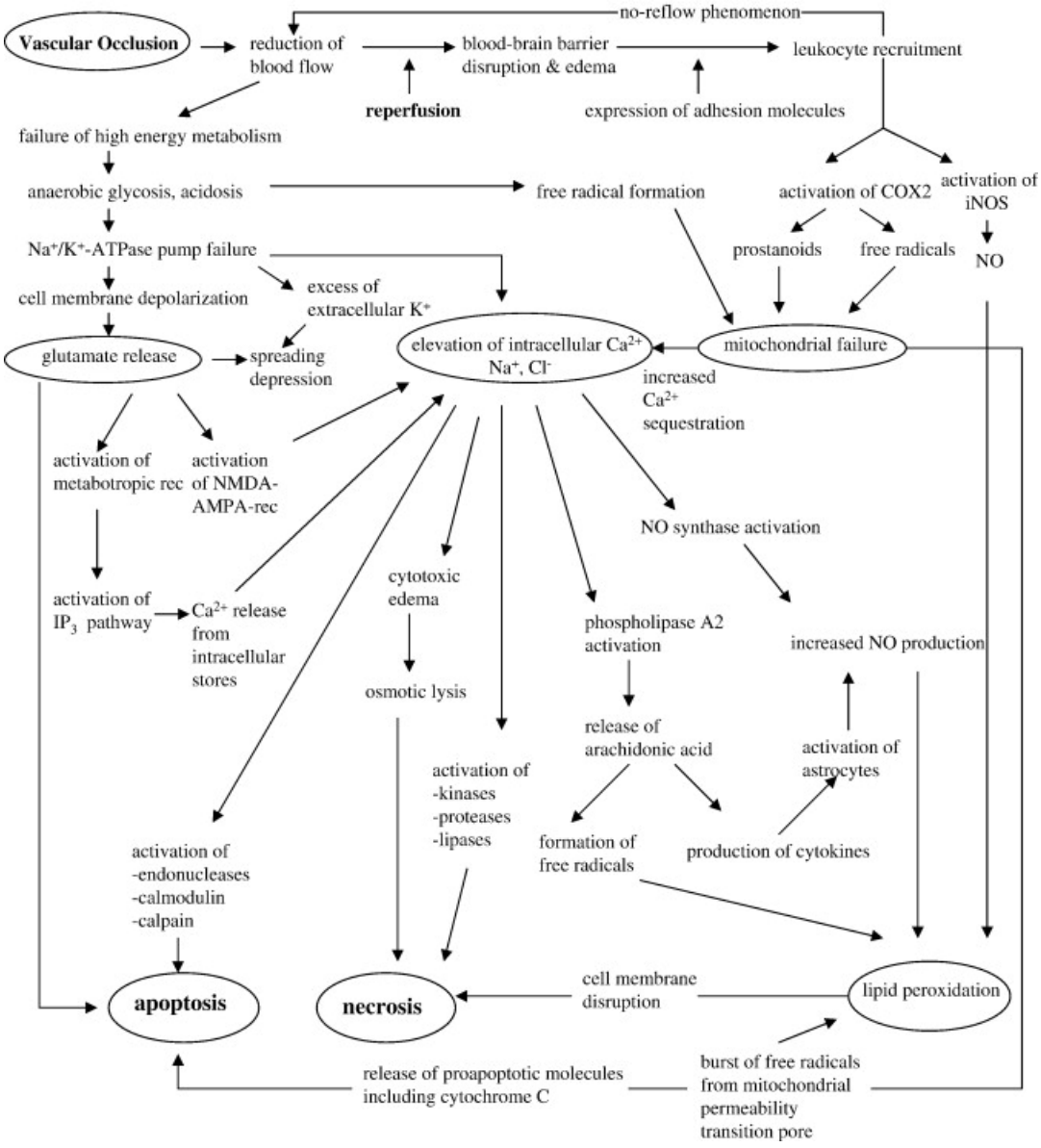
Post stroke epilepsy[15] is defined as " recurrent seizures following stroke with confirmed diagnosis of epilepsy". Epilepsy develops in about one third of early onset and half of late onset seizure[42.] It has been

observed that the diagnosis of epilepsy has considerable social and psychological impact on patients and it should not be made lightly. Even in patients with mild and well controlled epilepsy, self reported health related quality of life is significantly lower. Myint et[15] al observed mild degree of depression and anxiety in cases with post stroke seizures .

PATHOGENESIS OF SEIZURES IN STROKE:

Early and late onset seizures:

During acute ischaemia, the accumulation of intracellular sodium and calcium may result in depolarisation of the membrane potential. The local ionic shifts may lower the seizure threshold[(27)] . In experimental model, glutamate excitotoxicity is a well established mechanism of cell death which is shown below:



The size of regional metabolic dysfunction may also be relevant in the mechanism of early onset seizures. In the setting of a larger area of ischaemic hypoxia, excitotoxic neurotransmittess may be released extracellularly at high levels.

In studies of experimental animal models, neuronal population in the neocortex and hippocampus have altered membrane properties and

increased excitability which probably lower the threshold for the initiation of seizure activity. The ischaemic penumbra adjacent to the ischaemic are contain electrically irritable tissue that may be a focus for initiation of seizure activity.

Global hypoperfusion can also lead to seizure activity in addition to focal ischaemia. Hypoxic ischaemic encephalopathy is one of the most important cause of status epilepticus. Hippocampus is particularly vulnerable to ischaemic onset which is an epileptogenic area.

In contrast to early onset seizure, persistent changes in neuronal excitability occur in late onset seizures. Replacement of healthy parenchyma by neurologlia and immune cells may play a role. A gliotic scarring has been implicated as a nidus for seizures of late onset, just as meningocerebral cicatrix in late onset post traumatic epilepsy.

PATHOGENESIS OF SEIZURES IN HAEMORRHAGIC STROKE:

The mechanism of seizure initiation by intra cerebral haemorrhage is not well established. Metabolic products of blood such as haemosiderin may cause focal irritation leading to initiation of seizure activity as shown in animal models.

PATHOGENESIS OF SEIZURES IN EMBOLIC STROKE:

The concept of cardiogenic emboli causing acute seizure is controversial with very few supporting data as already described. Cardiac and large vessel embolus frequently lodge at distal cortical branches. The possible mechanism by which cortical lesion predisposing to initiation of seizure may include depolarization within the ischaemic penumbra, rapid reperfusion after distal migration and fragmentation of the emboli.

CAN WE PREDICT WHO IS GOING TO DEVELOP POST STROKE SEIZURE?

Certain factors are associated with higher incidence of post stroke seizures. In ischaemia, the severity of the neurological deficit, severity of persistent disability after stroke, larger the infarct size, infarct involving multiple sizes, cortical damage, and hippocampus involvement are the important factors associated with the likelihood of developing seizures after stroke. The presence of structural brain lesion, EEG abnormalities and occurrence of partial seizures also carry a higher recurrence rate.

Impact of seizure on the outcome of stroke:

Severity of stroke is the most important factor that determine the outcome of stroke. However early seizures originating in penumbral

areas might be harmful due to the additional metabolic stress to the already vulnerable tissue[42]. A prospective study on the frequency, characteristics and prognosis of epileptic seizures at the onset of stroke [12], found higher mortality (30.8%) at 48 hours among patients with early onset seizures than those who had late onset seizures 7.4%,($p<0.01$). In contrast, the SASS strongly found higher mortality rate among patients with seizures after 30 days and one year.

The mortality rate in stroke patients with status epilepticus is high. A prospective study by Waterhouse EJ et al[43] reported almost three fold increase in mortality rate among patients with ischaemic stroke and generalized status epilepticus as compared to patients with acute ischaemic stroke alone i.e. 39% versus 14% ($p<0.001$)

Diagnostic studies:

It has been described by Holmes[36] that patients with periodic lateralized epileptiform discharges(PLEDS) and independent bilateral PLEDS on EEG after stroke had the risk of development of seizures. Cortical involvement on neuroimaging is more predicitive of epilepsy than any single EEG finding. The absence of EEG abnormalities does not exclude cerebral ischaemia particularly in subcortical or subtentorial regions.

Uncommonly seizures may mimic ischaemia on neuroimaging.

Differential diagnosis:

The differential diagnosis of post stroke seizures may be due to other causes. Drug withdrawal(benzodiazepine), medications and metabolic disturbances e.g. glucose abnormalities typically cause generalized seizures or partial seizures in some. Migraine related focal phenomena and TIAs may mimic post stroke seizures and may also produce focal slowing on EEG findings.

Management of seizures in stroke:

Clinicians often face dilemmas whether to treat an isolated seizure and choice of anti epileptic during to be used in patients who developed single or recurrent seizures.

Several observational studies suggest that an isolated early seizures do not require treatment (or) can be very well controlled with a single drug[6,30] and patients developing recurrent early seizures or late onset seizure after stroke require pharmacological treatment. As per IAN guidelines,antiepileptic drug therapy for seizures need to be individualised.

Post stroke seizures are well controlled with a single anticonvulsant usually. In a retrospective study, seizures in 88% of patients were successfully managed with monotherapy[32]. Given the typical focal onset of seizures, the first line therapy options include carbamazepine and phenytoin sodium. Benzodiazepine-lorazepam should be initially administered intravenously in a patient with ongoing seizures.

In elderly patients, newer AEDS are being used as first line drugs because of their effectiveness and favourable profile of side effects. A trial with lamotrigine was recently demonstrated to be better tolerated and more effective in maintaining patients free of seizures for longer intervals than carbamazepine[44]. Recently gabapentin has been shown to be more effective as monotherapy for partial epilepsy[45]. But its major disadvantages are thrice a day dosing regimen and reduced clearance in renal disease. Other drugs studied as adjunctive agents are topiramate and levetiracetam.

In patients with intra cerebral haemorrhage and subarachnoid haemorrhage the Stroke Council of the American Heart Association recommended uniform seizure prophylaxis during the acute period[46].

The guidelines suggest phenytoin; with discontinuation of therapy after one month, if no seizure activity occur during the period. Patients with seizure activity more than 2 weeks after presentation have a higher

risk of recurrence of seizures and may require long term seizure prophylaxis.

Experimental studies showed that the use of phenytoin, phenobarbitone and benzodiazepine may impair post stroke recovery. But relevant clinical data are limited[48] .

As demonstrated in animal studies, antiepileptic drugs may also act as neuro- protectants.

Unanswered questions:

Several important questions regarding seizures in stroke need to be addressed in future research.

The current understanding of the epidemiology, pathophysiology and treatment of seizures in stroke remain incomplete.

Better definition of patients at high risk for the development of post stroke epilepsy remains unclear and such patients may benefit from therapies aimed at reducing epileptogenesis.

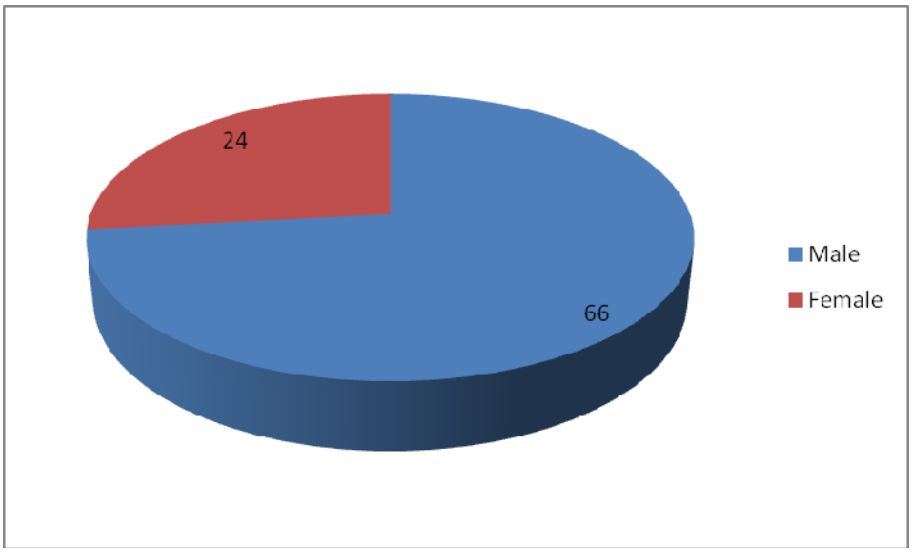
OBSERVATION, ANALYSIS AND RESULTS

Demographic profile:

Sex:

Of the 100 patients in the study group, there were 76 males, and 24 females.The sex ratio was male : female3.2:1

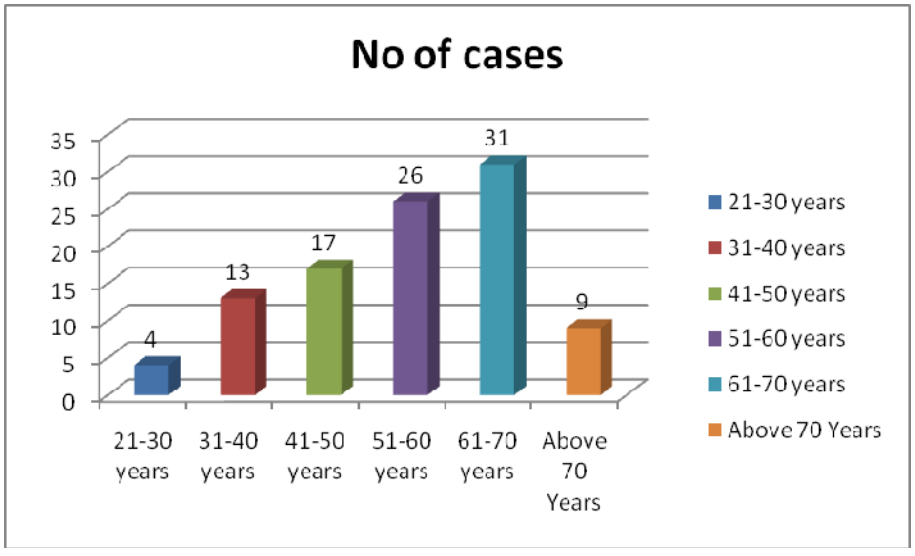
Gender	No of Cases
Male	76
Female	24



Seizures in relation to Age

The maximum number of patients (n=31) were from the age group (61 – 70yrs) showing that the elder age group prone for the development of stroke related seizures. The number of cases according to the age group was shown below:

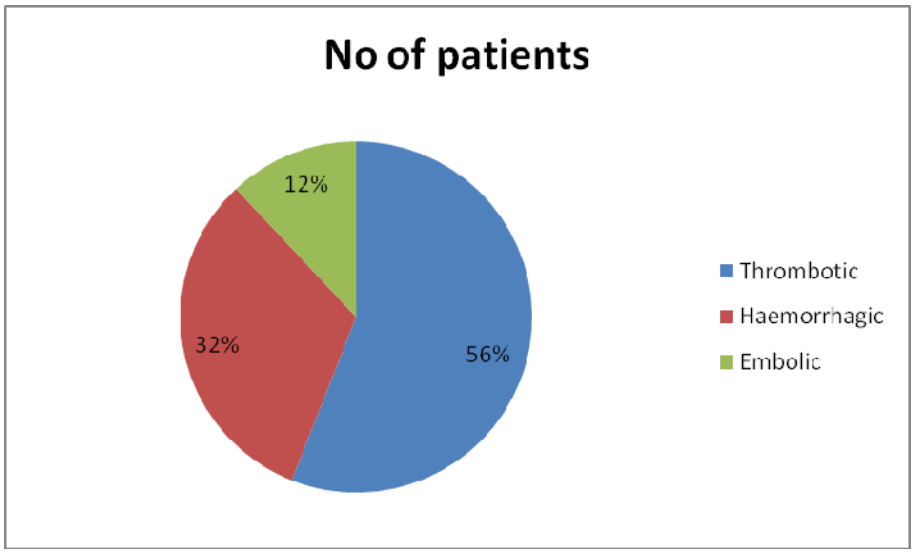
Age group in years	No of cases
21-30 years	4
31-40 years	13
41-50 years	17
51-60 years	26
61-70 years	31
Above 70 Years	9



Distribution of seizures according to stroke subtype:

Of the 100 patients, 68 had ischaemic stroke and 32 had intra cerebral haemorrhage as evidenced by the neuroimaging studies.

Of the 68 ischaemic stroke group, 12 had demonstrable cardiac source of emboli evidenced by the echocardiogram. Remaining 56 patients in the ischaemic stroke group were considered to have probable thrombotic etiology.



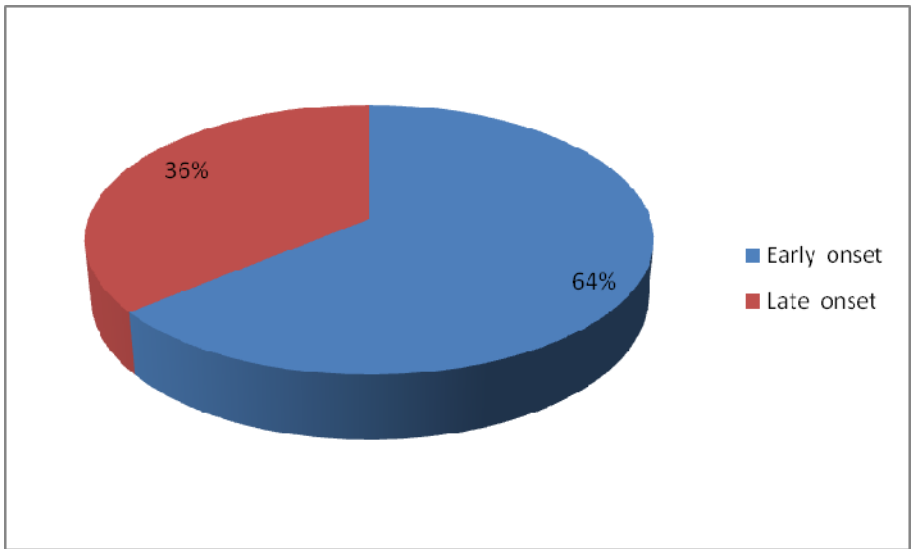
Distribution of seizures according to the timing and stroke subtype:

Early onset seizures were present in 64 patients and late onset in 36 patients. Of the 64 patients with early onset seizure, 50 patients (78%) had immediate onset of seizures (i.e) within the first 24 hours of stroke. Of the early onset group, cardioembolic stroke was present in 8,

thrombotic stroke was present in 36 patients. Intra cerebral haemorrhage was present in 20 patients. Of the late onset seizure group (n=36), 4 had cardioembolic stroke, 20 had thrombotic and 12 had intra cerebral haemorrhage.

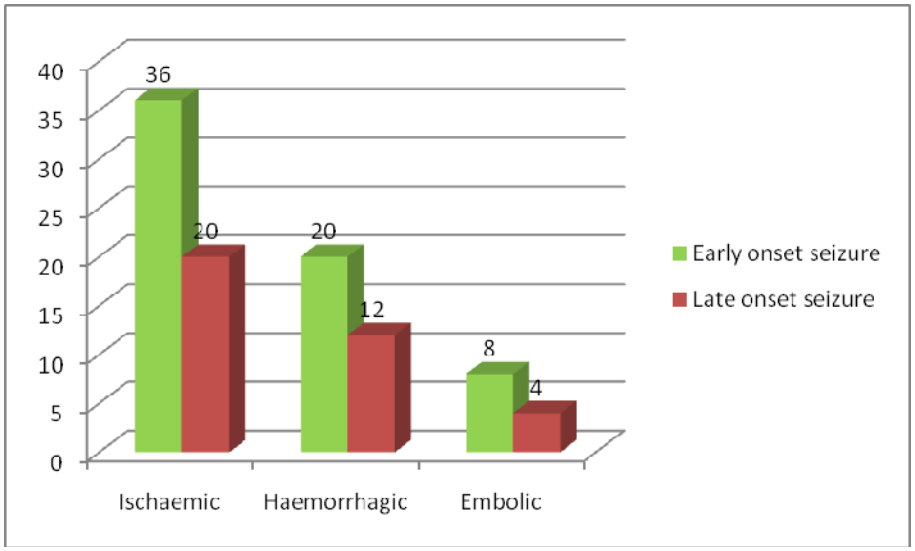
Timing of seizures

Timing of Seizures	No of Patients
Early onset	64
Late onset	36



Distribution of seizures according to the timing of seizures and stroke subtype

Type of stroke	Early onset seizure	Late onset seizure
Thrombotic	36	20
Haemorrhagic	20	12
Embolic	8	4

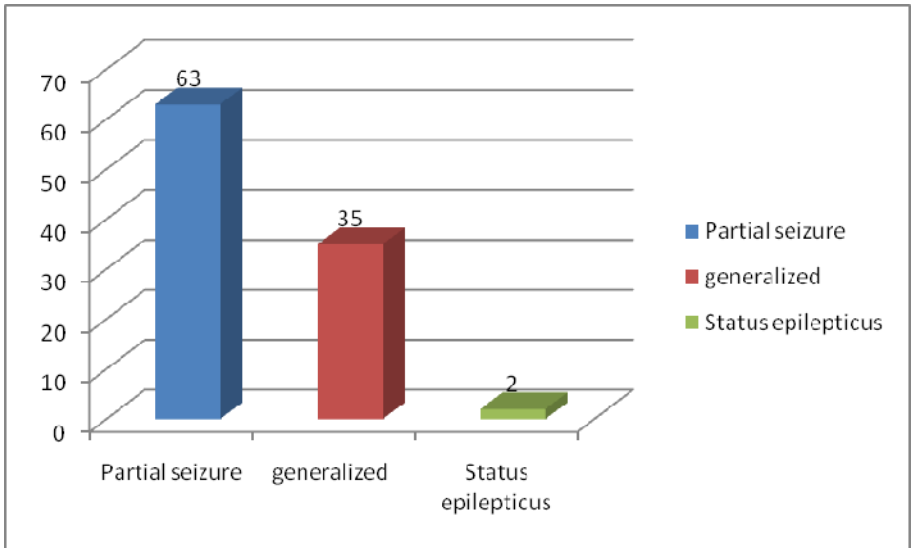


Seizure semiology associated with stroke:

In this study, 63 patients presented with partial seizure, 35 patients presented with generalized seizures and 2 with status epilepticus.

Seizure pattern associated with stroke

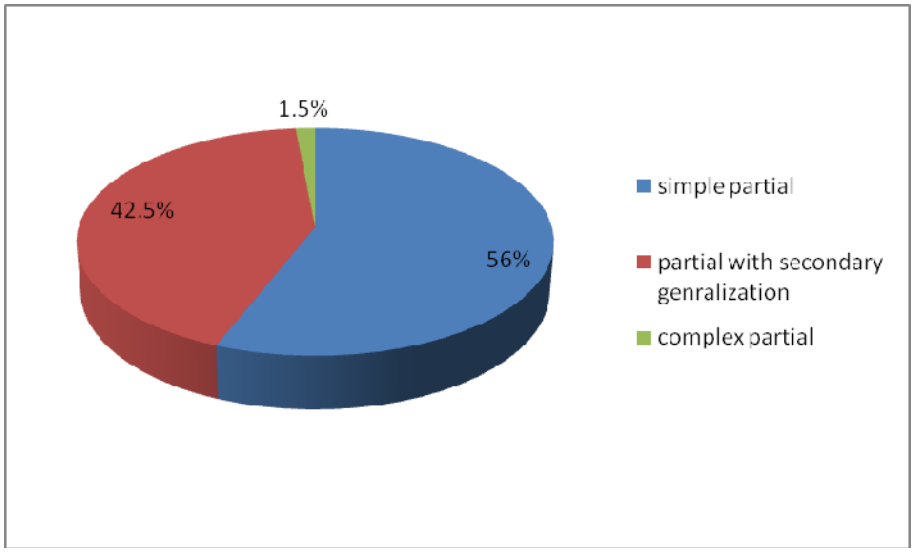
Seizure pattern	No of patients
Partial seizure	63
generalized	35
Status epilepticus	2



Partial seizure

Among the partial seizure subgroup (n=63), 35 had simple partial seizures (56%) and 27(42.5%) had partial seizure with secondary generalization. One patient had complex partial seizure (1.5%).

Type of seizure	**No of Patients**	%
Simple partial	35	56%
Partial with secondary generalization	27	42.5%
Complex partial	1	1.5%

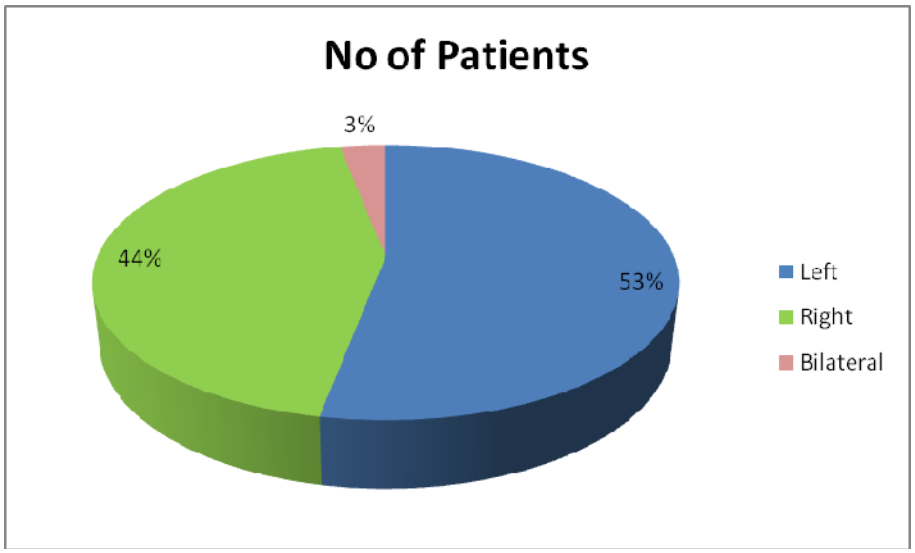


Anatomical site of stroke in relation to seizure:

Side of the lesion:

Of the 100 patients studied, predominant left sided (dominant side) lesion was present in 53 patients and right sided lesion in 44 patients. 3 persons had bilaterally predominant lesions as demonstrated by neuroimaging.

Side of lesion	**No of patients**
Left side	53
Right side	44
Bilateral	3



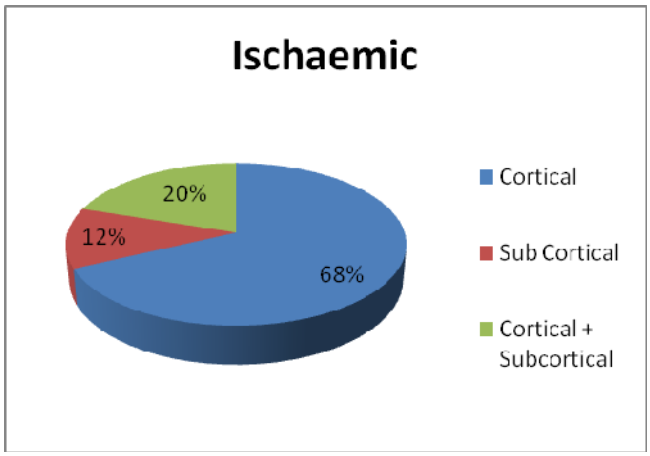
Depth of the lesion:

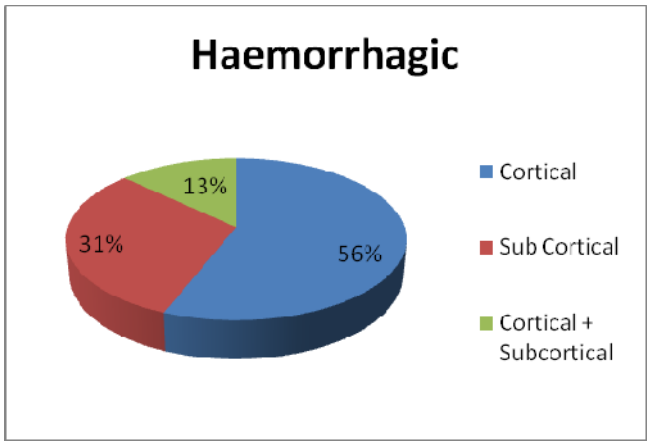
In the thrombotic group (n=56), 38 patients had cortical lesion (68%), 11 patients (20%) had both cortical and subcortical lesion and 7 (12%) had only subcortical lesions. Of the 32 patients with ICH, 18(56%) had only cortical involvement, 10(31%) had only subcortical involvement, 4 (13%) had both cortical and subcortical bleed.

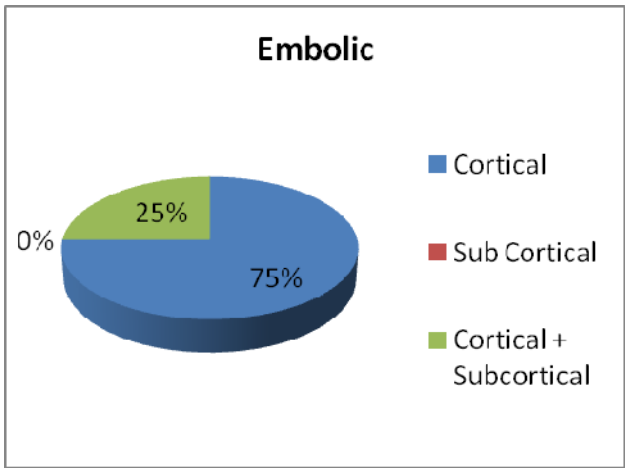
In the cardio embolic subgroup, 9(75%) had pure cortical involvement, 3(25%)had both cortical and subcortical involvement.

Anatomical site of stroke in relation to seizure

Type of Stroke	**Anatomical site**		
	Cortical	Sub Cortical	Cortical + Sub cortical
Ischaemic (56)	38 (68%)	7 (12%)	11(20%)
Haemorrhagic(32)	18(56%)	10(31%)	4(13%)
Embolic(12)	9(75%)	-	3(25%)


Ischaemic
20%
12%
68%
Cortical
Sub Cortical
Cortical + Subcortical


Haemorrhagic
13%
31%
56%
Cortical
Sub Cortical
Cortical + Subcortical


Embolic
25%
0%
75%
Cortical
Sub Cortical
Cortical + Subcortical

Size of the infarct:

Of the patients with ischaemic stroke (n=68), 42 patients (62%) had large infarcts and 26 patients(38%) had small infarcts. The lesion size was correlated with recurrent seizures.

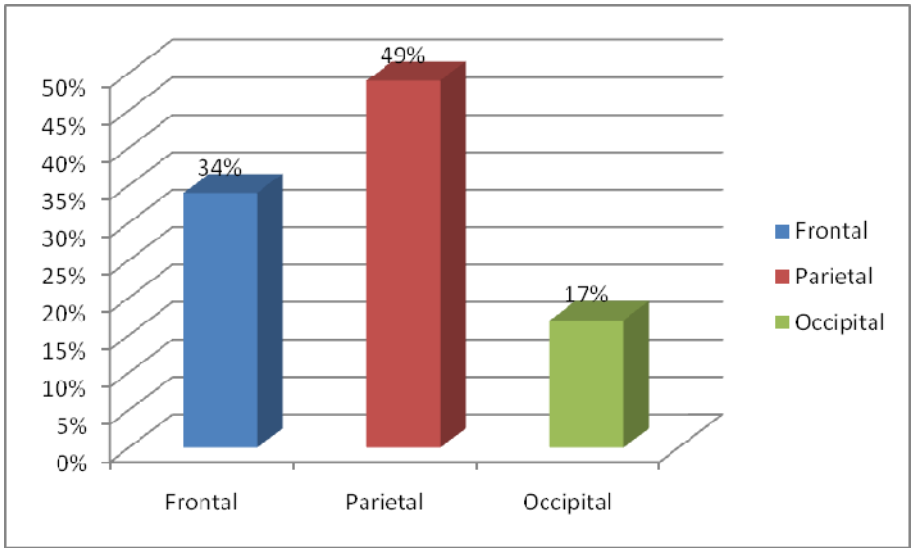
Lesion size	No of patients	%
Large >5 cm	42	62%
Small < 5cm	26	38%

Location of cortical infarcts:

Frontal sub group – 16(34%)

Parietal sub group – 23(49%)

Occipital sub group – 8(17%)

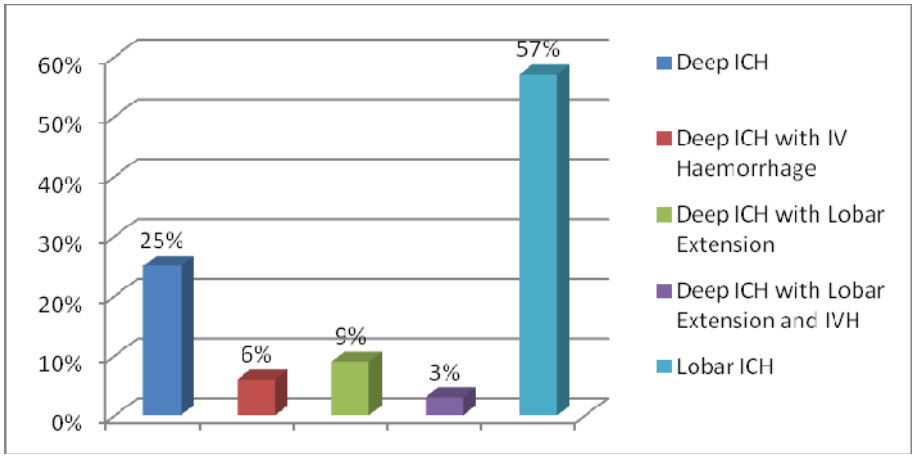


Of the total 47 (38 thrombotic, 9 embolic group) patients with pure cortical infarct, 23 (49%) had parietal infarct, 16 had (34%) frontal infarct, and 8 (17%) had occipital infarcts. The post stroke seizure was more common with the parietal group(49%) which includes parieto temporal, temporal and parieto occipital areas and least common with the occipital sub group.

Seizure in relation to intra cerebral haemorrhage:

As per the methodology, the patients were studied in relation to the deep ICH, deep ICH with intra ventricular extension with lobar extension, and with lobar ICH. The number of patients with the above categories of ICH is given below:

Site of lesion	No of patients (32)	%
Deep ICH	8	25
Deep ICH with IV haemorrhage	2	6
Deep ICH with lobar extension	3	9
Deep ICH with lobar extension and IVH	1	3
Lobar ICH	18	57



An applying Chi square trends

Chi square value : 7.12

p = 0.008, p = <0.05, highly significant

Volume of ICH

The patients with intra cerebral haemorrhage were classified as two subgroups according to the volume of blood and correlated with the seizure occurrence.

Volume	**No**
Small (0-20 ml)	20
Large (30ml or more)	12

EEG analysis:

Inter ictal EEG was done in 75 patients only. The EEG of all the patients presenting with seizures associated with stroke were categorized into 5 types as given below:

Type	**No of patients**
Type I – normal	15(20%)
Type II – diffuse slowing	7(9%)
Type III – focal slowing with / without diffuse slowing	36(48%)
Type IV – focal spikes, sharpwaves	15(20%)
Type V – presence of PLEDS	2(3%)

Stroke severity:

The data regarding stroke severity as assessed by Canadian Neurologic Score was as follows:

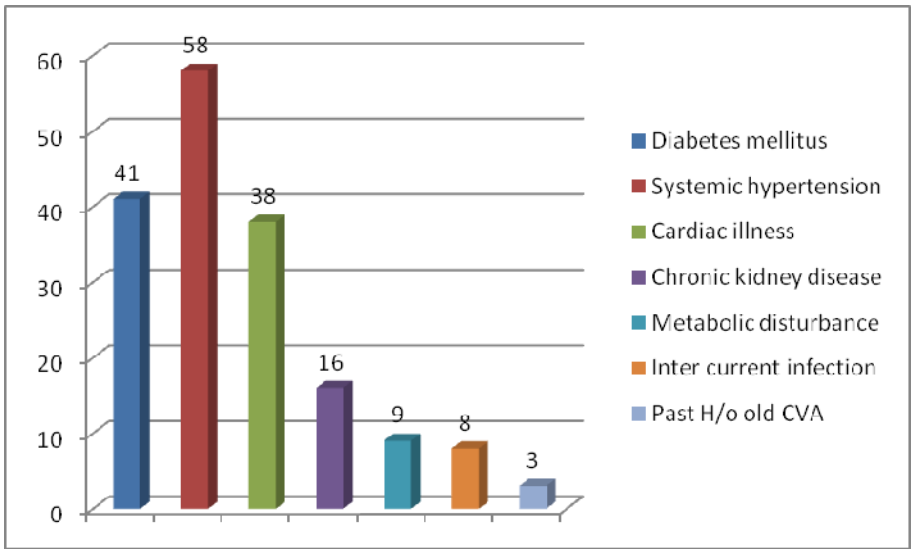
	CNS score	**No of patients**
Mild stroke	>7	34
Severe stroke	≤7	66

The stroke severity was correlated with the risk of seizures in stroke. The **mean CNS score in this study was 6.1**

Comorbid conditions:

The following comorbid conditions associated with seizures were studied

Comorbid conditions	**No of patients**
Diabetes mellitus	41
Systemic hypertension	58
Cardiac illness	38
Chronic kidney disease	16
Metabolic disturbance	9
Inter current infection	8
Past H/o old CVA	3



Out of the 41 patients with diabetes, 26 patients(63.4%) had a blood sugar of more than 200mg/dl at the time of admission. Out of the 58 patients with systemic hypertension, 42(72.4%) had a diastolic BP of more than 100.

The comorbid conditions were quantified by the Charlson – Deyo index. The score was more than 3 in 70 patients. Presence of the above comorbid conditions increase the risk of occurrence seizures in stroke.

Recurrent seizures after stroke:

In this study, seizures recurred in 40. Early onset seizure was associated with seizure recurrence in 14, late onset seizure in 26 with a **Chi square of 22.6 and p= 0.00004 which is highly significant.**

Type of seizures	**Total No**	**Recurrence no**	**Recurrence %**
Early onset	64	14	28%
Late onset	36	26	72%

The distribution of seizures as per the subtype of stroke is given below:

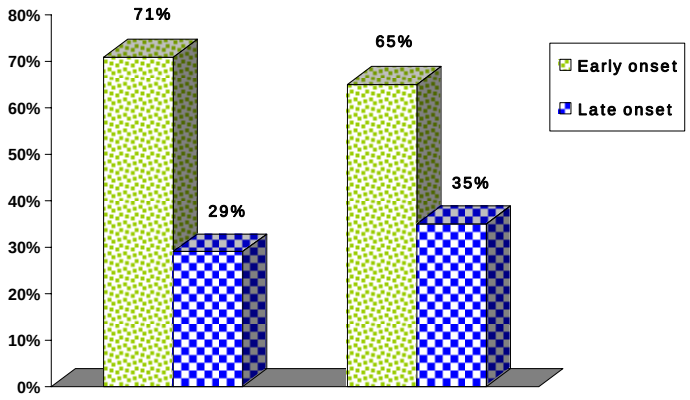
	Early onset seizure (n=64)		**Late onset seizure(n=36)**	
	No recurrence	Recurrence	No recurrence	Recurrence
Overall	50	14 (22%)	10	26(72%)
Ischaemic	36	10(71%)	5	17(65%)
Haemorrhagic	14	4(29%)	8	6(35%)

Early and late onset recurrence Chi square 22.3, p= 0.000004, p=<0.05 highly significant.

Early and late onset recurrence in relation to ischaemic and haemorrhagic type Chi square for linear trends 7.01, p= 0.008,p <0.05 significant

Late onset seizure in ischaemic stroke p=0.04, p <0.05 significant

Recurrence of seizures



Mortality:

Out of the 100 patients studied, and followed up over a minimum period of 6 months, 8 patients expired. Of the 8 expired, 6 patients expired in the early phase of the illness (i.e) within first week of illness. 2 patients expired in the later phase of follow up period.

Mortality (n=8)

Early death - 6

Late death - 2

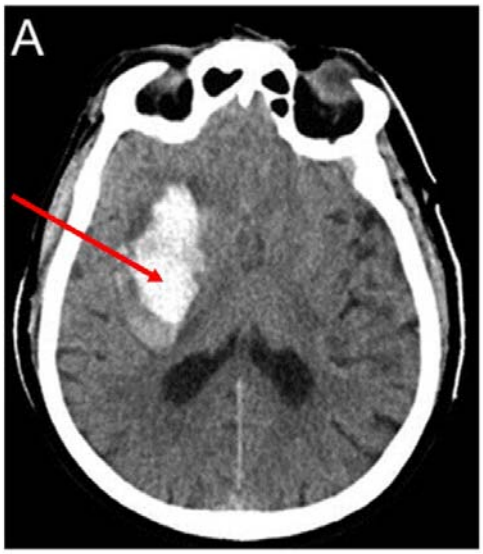
Of the 6 patients with early death, 3 had massive hemispheric infarct, 3 patients had massive ICH with intra ventricular extension with mass effect. 2 patients with ischaemic stroke died in the later phase of the

illness due to coexisted metabolic encephalopathy and respiratory infection.

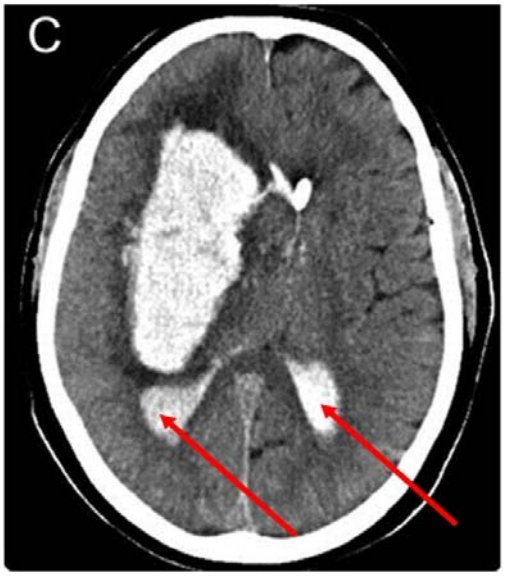
Sl.No	Death	Type of stroke	Comorbid status
1.	Early onset seizure	Massive hemispheric infarct	-
2.	Early onset seizure	Massive hemispheric infarct	Elevated blood sugar
3.	Early onset seizure	Massive hemispheric infarct	-
4.	Early onset seizure	Multiple infarct (embolic)	Atrial fibrillation / cardiac failure
5.	Early onset seizure	Massive ICH associated with midline shift	-
6.	Early onset seizure	Massive ICH associated with midline shift	Elevated renal parameters
7.	Late onset seizure	Ischaemic	Metabolic Encephalopathy
8.	Late onset seizure	Ischaemic	Respiratory infection

All the 8 patients died, had a Charlson – comorbid index of more than 3.

Rt Basal ganglia bleed



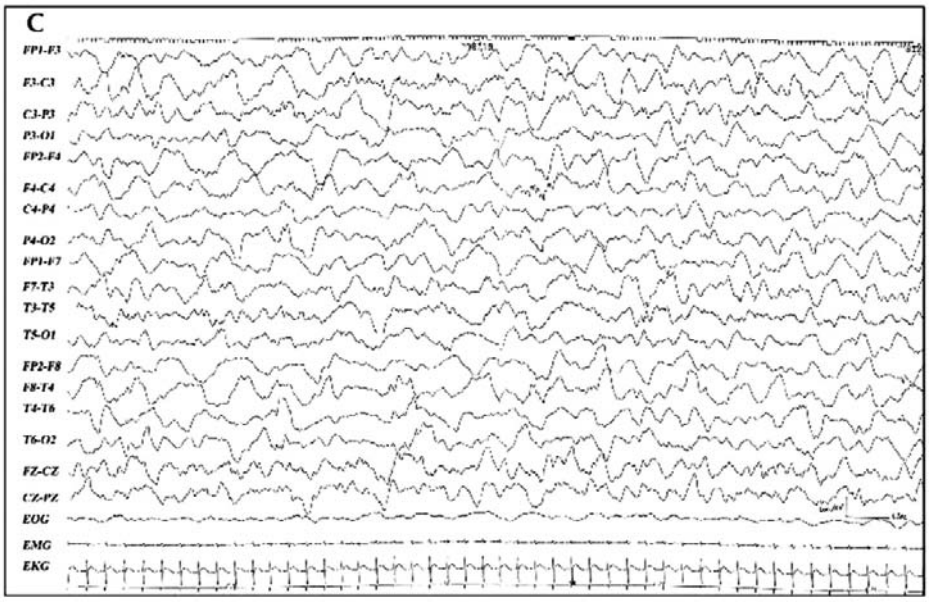
Rt Basal ganglia bleed with intraventricular extension



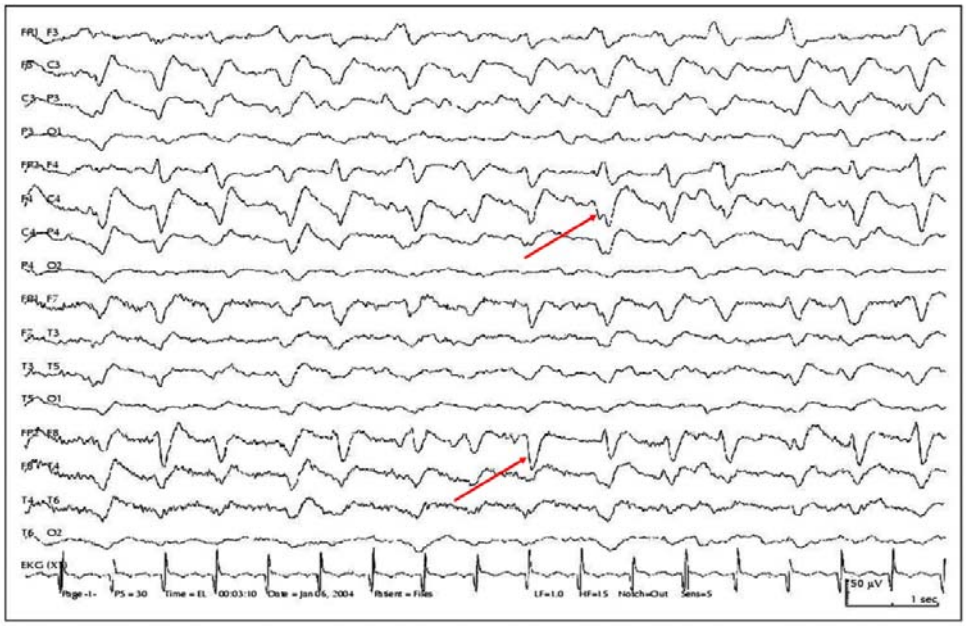
Rt Malignant MCA infarct (cortical & subcortical)

Lt Temporo parieto occipital infarct (MCA & PCA)

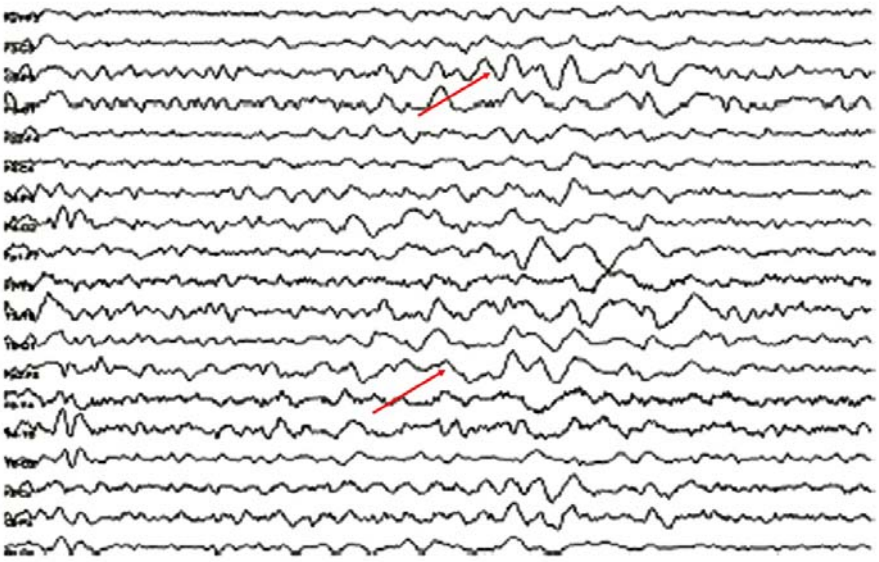
EEG Showing diffuse slowing



EEG Showing PLEDs



EEG Showing focal slowing over left temporal and parietal regions



DISCUSSION

One hundred patients presenting with seizures associated with arterial stroke were included in this study.

Age:

In this study maximum number of post stroke seizures (n=31)

occurred in the age group between 61-70. The study by Forsgren et al observed that stroke accounts for 30% of the newly diagnosed seizures in patients more than 60 years old. Hauser et al in his community based study conducted at Rochester ,USA also observed similar trend in the age specific incidence of post stroke seizures. So it is obvious from the present study and the above studies that the post stroke seizures are more common in the elder age group.

Site of the lesion: Right or Left

Few studies (Matsumura et al ,Gupta et al,Sitajeyalakshmi, NIMSHyderabad) observed that for reasons unknown, left hemispheric lesions are more prone to develop seizures after stroke than right hemispheric lesions. In this present study of seizures in stroke, left sided lesions (53%) are more common than left sided lesions (44%).

Type of stroke

Of the total 100 patients of seizures with stroke, 56 patients had ischaemic stroke as evident from the CT brain (or) MRI brain. 32 patients presented with haemorrhagic stroke, 12 patients had embolic stroke mostly cardioembolic as evidenced by echocardiogram. The incidence of seizures was 10.6 in patients with haemorrhagic stroke and 8.6 with ischaemic stroke as studied by Bladin et al [27].

Timing of seizure

Of the 100 patients presented with seizures associated with stroke, 64 had early onset seizures, 36 had late onset seizures. Of the 64 patients with early onset seizures, 50 patients(78%) had immediate onset of seizures (i.e) within 24 hours of stroke.

The frequency of early post ischaemic seizures in the largest studies[27,40,34,37,32] range from 21 to 33% , with 50 to 78% occurring within the first 24 hours after stroke.[(27,33,34)] The frequency of late post ischaemic stroke seizures varies from 3% to 67%[(27,33,40)]

Several studies quote that most early onset seizures occur during the first 1 to 2 days after ischaemia. About half (43%) of all patients in the Stroke After Seizures Study (SASS) experienced first seizure within the first 24 hours of stroke.[27] Most seizures associated with haemorrhagic

stroke also occur at onset or within first 24 hours as per the study by Romanik et al.[11]

In a study from India[9], the frequency of early onset seizures was 77% of which 2/3rd had immediate post stroke seizures. Out of the 97 patients five series[(49-52)] who had seizures in the post stroke period, seizures occurred in 55 at the onset of stroke.

The data of this present study also correlate well with the above results that there is a higher percentage of early onset seizures in both the ischaemic and haemorrhagic stroke.

On the contrary, in another series of 72 patients where only post ischaemic seizures were considered, only 24% of seizures were of early onset[(53)].

Considering haemorrhagic stroke, the incidence of early seizures tend to be higher than the late onset seizures as per the study by Sung Cchu et al[35]

Results of autopsy and clinical studies(So El et al, Krause JA et al, Richardson EP et al, Lesser RP et al, Mohr JP et al) suggest that early seizures are more common with cardioembolic stroke than with other types of stroke[34,62-5]. The present study also reported 58% of early onset seizures in embolic stroke group. However recent studies including

NINDS stroke data bank study[60] showed that there is no association between seizure at the onset of stroke and presence of cardiac source of embolism.

Semiology of seizures:

This study reported the semiology of seizures as follows - partial seizures 63%, generalised seizures 35% and status epilepticus – 2%. Of the partial seizure type, 56% had simple partial seizures, 42.5% had partial seizures with secondary generalisation. One patient(1.5%) reported complex partial seizure with a sense of fear as the presenting aura.

Several studies (Bladin CF et al, Lamy C et al,Kilpatrik CJ et al,Girous M et al) report approximately 50 to 90% of early onset seizures as simple partial seizures[27,37,38,39].

In contrast, the study of the prognostic value of early seizures in stroke by Arboix et al reported a higher frequency (50%) of generalised seizures without focal onset in patients with early onset seizures[40].

In a study of early onset seizures in 90 patients by Giroud et al[39], simple partial seizures were the most common type (61%) followed by focal onset seizures with secondary generalisation (28%).

In another series by Davalos et al and Gupta SR et al[31,32], early onset seizures were more likely to be partial and late onset seizures were more prone for secondary generalisation.

In an Indian study by Dhanuka et al[9] 78.6% of single initial seizure was focal, while 21.4% had generalised seizures. Focal seizures were the predominate type of seizures in early onset (74%) and late onset (75%) group.

On the contrary, in a study of post stroke seizures conducted at a tertiary care centre in Pakistan [10]reported 22% had partial and 78% had generalised seizures.The study concluded that generalised seizures are more common than partial seizures.

In a series of Susanna et al [53] early onset seizures were more likely to be generalised. Another study [11] reported the most common type of seizure as simple partial seizure, the rarest being complex partial seizure.

Stroke accounts for 25% of cases of status epilepticus in some series[41]. The present study reported 2% cases of status epilepticus. A single institutional study by Veligolu SK et al reported that 17 out of 1174 patients with ischaemic or haemorrhagic strokes (0.14%) developed status epilepticus[55]. Another second single institution study by Rumbach et al found that 22 of 2742 patients with ischaemic stroke (0.8%) had

status epilepticus[56]. In a large study of post stroke seizures 9% had status epilepticus.[55] It also observed that status epilepticus was not associated with higher mortality, stroke type (Ischaemic or haemorrhagic), topography (cortical involvement) or lesion size or EEG Patterns.

It is evident from all these studies and the present study except for very few studies, partial seizures are most common seizure type in stroke. Data regarding the seizure subtype varies with different studies. This is because several studies of post stroke seizure are limited by the retrospective design in majority of the studies and are potentially confounded by recall bias and interviewer bias related to obtaining the description of seizure from observers (or) patients. Upto 63% of seizures may not be recognized by patients[36]. Hence it is not surprising that different studies find varying frequencies of seizure subtype after stroke.

Lesion site and seizure association.

In this study in patients with ischaemic stroke, 68% had cortical infarcts, 20% had both cortical and subcortical infarcts. Only 12% had only subcortical infarcts. In patients with haemorrhagic stroke, 56% had cortical bleed, 31% had subcortical bleed with or without extension of edema into the cortical region and 13% had both cortical and subcortical Haemorrhage. In patients with embolic stroke 75% had cortical infarcts and 25% had cortical and subcortical infarcts. Hence it is evident from

the above data that cortical site is the more common cause for seizure in stroke.

Cortical location is the best characterised risk factor seizures after stroke and is supported by several studies. In multivariate analysis of data from the Seizures After Stroke Study (SASS) cortical location was a significant risk factor for stroke(HR - 2.09: 95% CI, 1.9-3.68,$p<0.01$). It also pointed out that the only risk factor for seizures after intracerebral Haemorrhage was cortical location(HR - 12.37; 95% CI;1.35 to 7.40, $p<0.008$).

However in a community based prospective study by Reith et al [57] the association between cortical involvement and post stroke seizures was not found.

Lobar site is considered to be the most epileptogenic in patients with intracerebral haemorrhage. The incidence of seizures was highest with bleeding into lobar cortical structures(54%) low with basal ganglia haemorrhage (9%) as shown by the study by Faught E Peters et al which included a series of 123 patients [30]. Caudate nucleus involvement and parietal or temporal lobe involvement within the cortex predicted the seizure[30]

The study by Dhanuka et al[9] observed that 85% of the post ischaemic seizures had cortical lesion with (or) without involvement of subcortical structures. 33% of patients with intracerebral haemorrhage had cortical haematomas while 80% of capsule ganglionic haematomas were large with extension of edema or haematoma to the cortical area. Only 14.28% of lesions (infarcts and haematomas) were localised to subcortical regions.

In the present study, 12% of patients with ischaemic stroke and 13% with haemorrhagic stroke had only subcortical lesions. Since MRI studies were not done in all patients in the present study, the possibility of cortical involvement still cannot be ruled out in these pure subcortical cases associated with seizures.

Size of the infarct:

Larger the size of the infarct, there is more risk for the development for post stroke seizures.This is evidenced by several studies(Lamy et al; Gupta et al). In this present study also, of the 66 patients with ischaemic stroke,42 patients(62%) had larger infarcts correlating well with the size of the infarct and occurrence of seizures

Location of the cortical infarcts:

In the present study, out of the cortical infarcts, parietal subgroup (which includes parieto temporal, temporal and parieto occipital areas) has the maximum number of patients with seizures after stroke(23 out of 47 patients with cortical stroke). The study by De Reuk et al showed that large cortical infarcts located in the parietal and temporal regions are associated with increased risk of seizures.However the study by Giroud et al observed that anterotemporal and posterofrontal ischaemic lesions are frequently associated with post stroke seizures.

Site of the intracerebral haemorrhage(ICH):

Lobar ICH was more prone for seizures as shown by many studies (Bladin et al,Dhanuka et al, Anupol et al). A Korean study by Kwang Moo et al observed that presence of cortical involvement, presence of intraventricular blood and development of hydrocephalus appear to be independent risk factors for developing seizures. In the present study also, except for the 8 patients with deep ICH(25%) and 2 with deep ICH and intraventricular extension(6%) , all the remaining 22 patients(69%) have lobar extension of haemorrhage- either primary lobar ICH(57%) or deep ICH with lobar extension(12%) correlating well with the above studies. Intraventricular extension of the haemorrhage was found in 9%.

Volume of intracerebral haemorrhage:

Several studies (Yue Chen et al, Dhanuka et al) observed that the volume of haematoma more than 30ml was associated with increased risk of seizures. In the present study, 18 out of the 32 patients(56%) with S large volume haematoma(30 ml or more). Even in patients with small volume haematoma(44%), two third had extension of edema into the cortex.

Recurrent Seizures

In this study recurrent seizures were present in 40% (22% with early onset seizures and 72% with late onset post stroke seizures).

A prospective study by Bladin et al reported that seizure recurrence was 55% patients with post stroke seizures[27] . Similar findings were observed in other studies also.

In another study,[10] Kheelani et al reported that the recurrent seizures were found in 21% at one year follow up. A study by Dhanuka et al[9] reported recurrent seizures in all the patients who had late onset seizures where as none of the early onset seizures developed recurrent seizures or epilepsy.

In a prospective study by Christopher F.Bladin et al [8] recurrent seizures occurred in 55% of post ischaemic stroke patients with late onset seizure. Recurrent seizures occurred in 100% of patients with late onset seizures after intracerebral haemorrhage. The study by De Reuck et al[16], reported that late onset seizures had a higher recurrence rate.

Susanna et al [53] reported recurrent seizures occur monthly in the late onset seizures group though few studies (Hauser et al, Kilpatrik et al) disagree[17,38.]

In contrast to the above mentioned studies and the present study, few studies favour early onset seizure after stroke as the risk factor for recurrent seizures. As evident from few studies, the early onset post stroke seizure is an independent risk factor for subsequent development of recurrent and late seizures.[(34,37)]Patients with early onset seizures were more likely to develop late onset seizures and approximately 16 times (95%CI, 5.5 to 40.2) more likely to develop epilepsy as confirmed with patents without early seizures.

Post stroke epilepsy is defined as recurrent seizures following stroke with confirmed diagnosis of epilepsy. Post stroke epilepsy develops in about 1/3[rd] of early onset and 50% of late onset seizure[58.] Multivariate analysis(27) showed that late onset post stroke seizures

were an independent risk factor for development of epilepsy(HR 12.37, 95% CI, 4.74 to 32.32; p<.001)

Another observation about recurrent seizures is that the seizure type. Most recurrent seizures were of the same type as the presenting episode of seizure[31,32]. The present study also had similar a result.

EEG ANALYSIS:

The commonest EEG abnormality observed in the present study was focal slowing(Type III) present in 48%. Similar correlation was observed in few studies(Gupta et al, Dhanuka et al). In this study, PLEDS were seen only in 3%. The study by Jaques De Reuck observed that PLEDS was present in 5.8% of the post stroke seizures. However the study by Yun Chen et al reported that PLEDS were not observed in any of the post stroke seizures.

Holmes et al [51] reported that, patients with PLEDs and B/L independent PLEDS on EEG after stroke were especially prone to development of seizures. The patients with focal spikes had a risk of 78%; focal slowing, diffuse slowing and normal findings in EEG were associated with relatively lower risks 20%, 10% and 5% for development of recurrent seizures respectively.

EEG findings were correlated with recurrent seizures(discussed later).

No specfic EEG pattern was associated with early versus late seizures or recurrent seizures in post stroke seizures as per the prospective study[9] by Dhanuka et al. So the author states that the prognostic value of EEG is of little importance.

Stroke severity:

The severity of stroke is positively associated with the risk of post stroke seizures as show by many studies (Bladin et al, Lamy et al). In this study also 66% had a CNS Score of less than 7 correlating well with the above studies.

Comorbid conditions:

In this study, out of the 100 patients, the Charlson – Deyo comorbid index score was more than 3 in 70 patients. Diabetes mellitus was present in 41 patients and systemic hypertension was present in 58 patients. Hyperglycaemic state (63.4%) and elevated blood pressure(72.4%) at the time of admission are more prone for the risk of the development of seizure as shown by this study. The study by Falco et al[64] observed that there was a significant correlation between hyperglycaemia and infarct size. Studies by Eleavon et al and Chen Y et

al also observed that the above mentioned comorbid conditions precipitated post stroke seizures. However the study by Burneo et al observed that there was no difference in the Charlson Deyo index between the post stroke seizure group and the control group.

Mortality:

Mortality rate is early seizures associated with stroke is higher than stroke without seizures. The study by Shinton et al reported a higher mortality at 48 hours among patients with early seizures (30.8%) versus those without early seizures. The study by Arboix et al also reported that early seizures were associated with increased in hospital mortality. In the present study also, early seizure was associated with more number of deaths i.e. 6 out of the total 8 deaths.

On analysis of the death cases, it was observed that of the 6 early deaths 3 had massive hemispheric infarcts; one had multiple infarcts associated with atrial fibrillation and cardiac failure. In the haemorrhagic stroke group, both of the 2 patients died had massive intra cerebral haemorrhage associated with midline shift and mass effect. The associated comorbid conditions were also observed and shown below. In all the 8 patients died, the Charlson comorbid index was more than 3.

The main causes of early deaths as discussed by many studies(Burneo et al, Somsak et al,Thailand, Danilo et al) are the severity of stroke – massive infarcts, massive ICH with mass effect and associated metabolic abnormalities. Early epileptiform activity has a deleterious effect on the infracted areas, perhaps by increasing the metabolic demand in a hypoxic tissue causing secondary brain damage. Cerebral blood flow, glucose and oxygen consumption increase substantially during seizure activity and still more if associated comorbidities are present to meet the tissue's increased requirements for energy(Burneo et al).

Status epilepticus and mortality :

The association between status epilepticus and mortality is unpredicted with various studies reporting varying results. In the study by Water house EJ[37] reported the presence of status epilepticus is associated with 3 fold increase in the mortality rate in stroke. However a study by Veligolu SK[55], a study of status epilepticus after stroke found no independent relationship between the mortality and occurrence of status epilepticus. The present study favours the latter study that all the 2 patients reported with status epilepticus, survived.

Analysis of recurrent seizures after stroke

In this study recurrent seizures were reported in 40 patients. Recurrent seizures were more common in the late onset group(72%) than in the early onset group(22%) with Chi square 22.3; p=0.00004 which highly significant. Also, recurrent seizures were more common with the ischaemic stroke compared with haemorrhagic stroke with a P value of 0.008 which is highly significant.

The study by Dodge et al[16] reported that 46% of all post stroke seizures recurred and of these recurrent seizures, initial seizures were of early onset in 50%, late onset in 33% and in 17%, the exact time could not be determined. Louis and MC Danel[15] reported that 81% of late onset seizures and 6% of early onset seizures recurred with an overall incidence of 40%. Contrary to this, in the study by Gupta et al[39], there was no difference in the incidence of recurrence with regard to the onset of initial seizures.

So in the present study it was observed that recurrent seizures were more common in the late onset seizure with ischaemic stroke with a p value of 0.004 which is highly significant.

EEG and recurrent seizures:

In the present study the majority of the patients with recurrent stroke had type III abnormality (i.e.) focal slowing with / without diffuse slowing. All the patients with PLEDs(2) had recurrent seizures.

EEG abnormality	No of patients	Recurrence
I	15	7
II	7	5
III	36	18
IV	15	8
V	2	2

In the study by Gupta et al, higher incidence of recurrent seizures was present in patients with Type II (75%) abnormality and PLEDS (100%). The higher incidence of recurrent seizures in these two abnormalities is explained by the fact that these two types (II, V) EEG abnormalities indicate a larger area of involvement of brain than the other types.

Comorbid conditions and seizure recurrence:

Presence of comorbid conditions and associated metabolic abnormalities precipitate seizures in many.

In the present study of the 40 patients with recurrent seizures, 14 patients had elevated blood sugar and 2 patients had hyponatremia.

Several authors (Anne pol et al, Burneo et al, Gupta et al) studied the association of comorbidities in post stroke seizures and came out with varying results. A Canadian multicentre cohort study observed no differences in the scores for the Charlson Deyo index between post stroke seizure group and non seizure group.

However the clinical study of post infarction seizures by Gupta and Haheedy observed a precipitating factor in 86% of patients.

AEDs for Post Stroke Seizures

In this present study all the patients were prescribed AEDs whether early or late onset. In patients presenting with recurrent seizures, 60% admit that poor compliance of the drug as the cause for recurrence. Other possible reasons for poor drug efficacy could be, drug interaction in patients taking cardiac drugs or oral anticoagulants which interfere with the metabolism of AEDs and decreasing the therapeutic levels .

Several observational studies suggest than an isolated early seizure after stroke do not require treatment (or) can be controlled easily with a single [54,12,59] drug.

However the study by Hauser Annegers[17] reported atleast one seizure relapse in 50% of patients who received AED after the first seizure after stroke during follow up period of forty seven months.The present study is limited by the shorter follow up period i.e., 6 months.

The study by Gilad et al[60] reported that beginning of treatment after the first early seizure after stroke was not associated with reduction of recurrent seizures after discontinuing the medication. In a retrospective study by SR Gupta seizures in 88% could be managed with monotherapy[32.]

The first line therapy option for post stroke seizures include carbamazepine and phenytoin. However the newer AEDs has been tried as first line agents for elderly patients[19]. Gabapentin has been shown to be efficacious[45] for partial seizures. A recent trial with lamotrigine demonstrated better tolerability and maintain longer seizure free intervals than carbamazepine.[20] Newer anti convulsants topiramate[21], levetiracetam[61] have been studied as monotherapy and as adjunctive agents for refractory seizures with variable results.

CONCLUSION

1. Partial seizures are common in stroke related seizures than GTCS, CPS being the rarest presentation.
2. Early onset seizure (seizures within 2 weeks of stroke) is the most common type of stroke related seizure.
3. Seizures are common in

   a). cortical lesions than with isolated subcortical lesions.

   b). Left sided lesions than with right sided lesions.

4. In patients with ischaemic stoke, seizures are more common with large infarcts (infarct size >5cm) and in parietal subgroup which includes a major portion of the temporal lobe.
5. In haemorrhagic stroke, lobar ICH and large volume ICH (>30 ml) have the risk for developing post stroke seizures.
6. The factors contributing to recurrence in post stroke seizures include late onset seizures, post ischaemic seizures, presence of PLEDS in EEG and poor AED compliance.
7. Associated comorbid conditions also play a role in stroke related seizures.
8. Mortality related to post stroke seizures is higher in early onset seizure than in late onset seizure.

SUMMARY

Despite the relatively lower incidence of seizures after stroke, post stroke seizure is one of the most common causes of epilepsy due to high incidence of stroke. Much additional work is needed to better understand the social impact of post stroke seizures, their prevention and effective management. Areas of future research regarding seizures in stroke include assessing the delayed patient outcomes and developing newer antiepileptic drugs with more neuroprotective effects. Post stroke seizure may also become a basic model in research that aims to prevent the injured cerebral cells into an epileptic focus.

BIBLIOGRAPHY

1.Adams and Victor's Principles of Neurology; Ninth Edition.

2.Neurology in Clinical Practice; Walter G. Bradley; Sixth Edition

3.Caplan's Stroke- A Clinical approach;Fourth Edition

4.Stroke syndromes;Julian Bogousslavsky,R.Caplan, second Edition

5.Uncommon causes of stroke; Julian Bogousslavsky,R.Caplan,First Edition

6.Oswald Camilo, Seizures after Ischaemic Stroke;Stroke 2004;35:1768-1775

7.Isaac E.Silverman M.D.,Post stroke Seizures; Arch Neurol, Feb.2002,vol59,No.2

8.Christopher F.Bladin,M.D. FRACP; Seizures After Stroke, Arch. Neurol.2000;57(11):1617-1622.

9.Dhanuka AK, et al, Seizures after stroke;Neurol India;2001:vol 49, page 33-6.

10.Kheelani BA,Post Stroke Seizures: J Pak Med Assoc.2008 July; 58(7):365-8

11.Romaiak A,Post Stroke Epilepsy,Neurol Neurochir Pol. 1998 May-Jun: 32(3):603-13

12.Shinton et al, Frequency, characteristics of seizures at the onset of stroke; J Neurol Neurosur Psych 1988: 51:274-276.

13.P.M.Vespa, Acute Seizures after intracerebral haemorrhage; Neurology, May13,2003: vol.60, no.9;1441-1446.

14.Reith, Seizures in stroke:Predictors and prognosis stroke 1997;28:1585-1589.

15.Myint,Post stroke seizure ; Postgrad.Med.J. 2006 Sept. 82(971)569-572.

16.De Reuck JL, Stroke related seizures and epilepsy;Neurol Neurochirn Pol. 2007 Mar-Apr;41(2):144-9.

17.Hauser W Annegers, Incidence of epilepsy and unprovoked seizures; Epilepsia 1993:34:453-468.

18.Bladin CF,Johnston et al; What causes seizures after stroke? Stroke 1994;25:245.

19.Cloyd J, Antiepileptics in the elderly: Fam Med.1994; 3:590-597.

20.Brodie Stall; UK Lamotrigine Study Group , double blind randomised comparison between lamotrigine and CBZ in the elderly with epilepsy. Epilepsy Res.1999;38:81-86.

21.Privitera, Topiramate YE Study Group in refractory partial epilepsy. Neurology 1996; 46:1678-1683.

22.Richardson EP, Epilepsy in cerebrovascular disease;Epilepsia 1954:3:49-65.

23.Aring CD, Differential Diagnosis between ICH and thrombosis, Arch Int.Med.1935;56:436-56.

24.Caplan LR, Intracerebral Haemorrhage . Boston:Butterworth-Heinemann, 1994, pp31-43.

25.Bladin CF, Seizures after stroke. M.D. Thesis, University of Melbourne,Australia 1997.

26.Heuts-Van:Seizures following a first cerebral infarct,Thesis.Rijksuniversiteet, Limberg, Maastricht Netherlands 1996.

27.Bladin CF, Seizures After Stroke Study(SASS): a prospective multicentre study. Arch.Neurol.2000;57:1617-1622.

28.Bogousslavsky; The Lausanne Stroke Registry: Analysis of consecutive patients witht stroke;Stroke 1998; 19: 1084-1092.

29.Kittner SJ, Cerebral infarction with a cardiac source of embolism in the NINCDS Stroke Data Bank: Neurol1990;40: 282-284.

30.Faught E.; Seizures after intracerebral haemorrhage. Neurol 1989;39:1090-1093.

31.Davalos et al; Seizures at the onset of CVA; Cerebrovasc. Diseases 1992;2;328-331

32.Gupta SR; Post Infarction Seizures , a prospective study.Stroke 1988;19:1478-1481.

33.Burn J, Epileptic Seizures after stroke: the Oxfordshire Community Stroke Project. BMJ 1997;315:1592-1587.

34.So EL, A study of seizure disorders after cerebral infarct. Neurol,1996;47:350-355.

35.Sung Cchu; Epileptic seizures in ICH. J Neurol Neurosurg Psych 1989; 52:1274-1276.

36.Blum DE, Awareness of seizures. Neurology. 1996;47:261-264.

37.Lamy C, Early and late seizures after stroke in young adults. Neurol.2003;60:401-404.

38.Kilpatrick CJ,Epileptic seizures in cerbro vascular accident . Arch. Neurol.1990;47:156-160.

39.Giroud M, Early seizures after acute stroke: a study of 1640 cases.Epilepsia 1994:36:959-965.

40.Arboix A, Prognostic value of early seizures in stroke; Eur Neurol 2003; 50:79-84.

41.Afsar N, Stoke and status epilepticus: Seizure.2003;16: 23-27.

42.Oslen T.S. Post stroke Seizures.Curr.Atherosclero. Rep. 2001. 3340-343.

43.Waterhouse , Effect of status epilepticus and cerbrovascular ischaemia on mortality.Epilepsy 1998; 29:176-183.

44.Chadwick, A double blind controlled trial of gabapentin monotherapy for partial seizures. Neurology.1998;51:1283-1288.

46.Broderick, Management of intracerebral bleed ,guidelines. Stroke 1999,30:904-915.

47.Qureshi, Spontaneous ICH, NEJM 2001; 344:1451-1460.

48.Goldstein,Recovery after stroke- influence of drugs. J Neuro Rehab.1990;4:138-144.

49.Louss, Seizures in non embolic cerebral infarction. Arch Neurol.1968;17:415-418.

50.Richardson EP, Incidence and pattern of seizures in autopsy proven cases of CVA.Epilepsia 1954;3:48-65.

51.Homes GL, EEG as a predictor for seizures in cerebral infarct and haemorrhage. Epilepsia 1954 ; 3:49-65.

52.Cocito L, Seizuress in cerebrovascular disease.Stroke 1982;13:189-195.

53.Susanna et al, EEG, CT and neuro sonographic findings in patient with post ischaemic seizures. J Neuro Sci.1995;132: 58-60.

54.De Carolis P, Late seizures in cerebrovascular occlusive disease . J Neurol Neurosurg Psychiatry.1984;47:1346-8.

55.Velioglu SK, Status epilepticus after stroke. Stroke 2001;32:1168-1172.

56.Rumbach L, Status epilepticus in stroke. Neurology.2ooo;54:351-354.

57.Reith J, Seizures in stroke, The Copenhagen Study. Stroke 1997;28.

58.NaeesH, Longterm outcome of cerebral infarct in the young . Arch Neurol Scand. 2004 1107-112.112.

59.Lo YK, Yiu Ch, Frequency and characteristics of seizures in Chinese acute stroke. Acta Neurol Scand. 1994;90:84-85.

60.Gilad R, AEDs in early post ischaemic stroke seizures: Cerebrovasc. Dis.2001;12:38-43.

61.Cereghino, Levetiracetam for partial seizures. Neurol2000;55:237-242.

62.Kwang Woo Moo et al, Seizures after spontaneous ICH, J Koreal Neuro Soc. Oct2012;52(4): 312-319

63.S.Sitajayalakshmi et al,NIMS, Hyderabad, India,Post Stroke Epilepsy, Neurology India,Vol 50. Dec.2002,pp.s79-s84.

64.Usha Misra and Kalita, Management of provoked seizures , Ann Ind Acad Neurol 2011 Jan-Mar, 14(1):2-7

PROFORMA

A STUDY OF SEIZURE IN STROKE

Name: Age/Sex: Place: D.O.A:

Death: Yes/No

HISTORY:

Seizures: Generalised / focal / Status

No of episodes:

Aura:

Relation to CVA – Onset

Immediate

Early

Late

Recurrence of seizures:

Details of AED intake:

Others:

CVA: Onset –

Duration:

LOC/aphasia/weakness – Rt / Lt / Others:

Past History: SHT / DM / CAHD / drug intake / others

General Examination: Pulse: BP:

EXAMINATION OF NERVOUS SYSTEM:

Higher functions:

Cranial Nerves:

Spinomotor system:

Sensory system:

Cerebellum:

EPS / ANS:

Canadian Neurological scale (CNS):

Charlson Deyo Index:

INVESTIGATIONS

Blood: Sugar mg/dl / Urea mg/dl S.Creatinine mg/dl

S. Electrolytes: meq/L

Neuro imaging:

CT Brain:

MRI Brain:

EEG:

Others:

Follow up:

Recurrent seizures:

Section A2	Motor Functions	Weakness	Score
COMPREHENSION DEFICIT		Symmetrical	
	Face Arms Legs	Asymmetrical Equal Unequal Equal Unequal	0.5 0.0 1.5 0.0 1.5 0.0

TOTAL: _____

References

CANADIAN NEUROLOGICAL SCALE

Patient Name: _____

Rater Name: _____

Date: _____

Mentation Score

Provided by the Internet Stroke Center — www.strokecenter.org

Cote, R, Hachinski, V. C., Shurvell, B. L., Norris, J. W., and Wolfson, C. "The Canadian Neurological: Scale A preliminary study in acute stroke."
***Stroke* 1986; 17:731-737**

Cote R, Battista RN, Wolfson C, Boucher J, Adam J, and Hachinski VC. "The Canadian Neurological Scale: Validation and reliability assessment."
***Neurology* 1989; 39:638-643**

Cheryl D. Bushnell, MD; Dean CC, Johnston, FRCPC; Larry B. Goldstein, MD. "Retrospective Assessment of Initial Stroke Severity. Comparison of the NIH Stroke Scale and the Canadian Neurological Scale."

Level Consciousness Orientation Speech	Alert Drowsy Oriented Disoriented/NA Normal Expressive Deficit Receptive Deficit	3.0 1.5 1.0 0.0 1.0 0.5 0.0
		TOTAL: _____

Section A1	Motor Functions	Weakness	Score
NO COMPREHENSION DEFICIT	**Face Arm: Proximal Arm: Distal Leg: Proximal Leg: Distal**	None Present None Mild Significant Total None Mild Significant Total None Mild Significant Total None Mild Significant Total	0.5 0.0 1.5 1.0 0.5 0 1.5 1.0 0.5 0 1.5 1.0 0.5 0 1.5 1.0 0.5 0
			TOTAL: _____

Charlson/Deyo Score

Submitted by acsadmin on Fri, 2009-10-09 13:29

DD_category:
PATIENT DEMOGRAPHICS

PUF Data Item Name:

CDCC_TOTAL length: 1 Allowable values: 0-2 Description:
Comorbid conditions as described by Charlson/Deyo (1992) are mapped from as many as ten reported ICD-9-CM secondary diagnosis codes reported for cases diagnosed January 1, 2003 and later. A single summary cumulative value is represented.
Analytic Note:
Because of the small proportion of cases with a Charlson Comorbidity score exceeding 2, the data have been truncated to 0, 1, 2 (greater than 1). A score of 0 indicates "no comorbid conditions recorded".

Charlson Comorbidity Score Mapping:

Reported ICD-9 CM	Codes Condition	Charlson Score
410 – 410.9	Myocardial Infarction	1
428 – 428.9	Congestive Heart Failure	1
433.9, 441 – 441.9,785.4, V43.4	Peripheral Vascular Disease	1
430 – 438	Cerebrovascular Disease	1
290 – 290.9	Dementia	1
490 – 496, 500 – 505, 506.4	Chronic Pulmonary Disease	1
710.0, 710.1, 710.4, 714.0 – 714.2, 714.81, 725	Rheumatologic Disease	1
531 – 534.9	Peptic Ulcer Disease	1
571.2, 571.5, 571.6, 571.4 – 571.49	Mild Liver Disease	1
250 – 250.3, 250.7	Diabetes	1
250.4 – 250.6	Diabetes with Chronic Complications	2
344.1, 342 – 342.9	Hemiplegia or Paraplegia	2
582 – 582.9, 583 – 583.7, 585, 586, 588 – 588.9	Renal Disease	2
572.2 – 572.8	Moderate or Severe Liver Disease	3
042 – 044.9	AIDS	6

‹ Great Circle Distance up Sequence Number ›

Sl.No	AGE / SEX	CVA (Side)	TYPE OF STROKE			Seizure Semiology	ONSET OF SEIZURE			Recurr	Choice of AED	ANATOMY			INFARCT			SIZE OF INFARCT		INTRA CEREBRAL HAEMORRHAGE				VOLUME OF ICH	EEG	Comorbid condition	CNS Score	Charlson Index	Death
			Thromb	Hgic	Emb		IMM	Early	Late			Cortical	Subcortical	Cortical + Subcortical	Frontal	Parietal	Occipital	Small	Large	Deep ICH + IVH	Deep ICH +Lobar	Deep ICH + IVH + Lobar	Lobar	Small / Large					
1	45/M	Lt	+	-	-	Partial	+	-	-			+	-	-	-	+	-	-	+	-		-	-	-	I	HT	5	1	
2	60/M	Rt	+	-	-	Partial	+			+	CBZ	+	-	-	-	+	-	+	-	-		-	-	-	III	HT,DM	6.5	>3	
3	70/M	Rt	-	+	-	GTCS	+	-	-	-	-	-	+	-	-	-	-				+		-	S	IV	CAD,HT	7.5	>3	
4	23/F	Lt	+	-	-	Partial/ gen	+	-	-	+	CBZ	+	-	-	+	-	-	-	+	-		-	-	-	III	HT	4	1	
5	40/M	Lt	-	+	-	Partial	-	-	+	+	CBZ	+	-	-	-	-	-			-		-	+	L	IV	HT,DM,CAD	8	>3	
6	30/F	Lt	+	-	-	Status	+	-	-	+	CBZ	+	-	-	-	-	+		+						III		2.5	>3	
7	75/M	Lt	-	+	-	GTCS	+	-	-		PHT	+	-	-			-			-		-	+	S	I	HT,DM	5.5	>3	Early
8	65/M	Rt	-	+	-	GTCS	-	+	-	+	PHT	-	+	-	-	-	-					+		S	III	HT,DM	10.5	>3	
9	65/M	Rt	+	-	-	Partial		+	-	-	CBZ	+	-	-	-	+	-	+							IV		7	0	
10	56/M	Rt	+	-	-	GTCS	+	-	-	+	CBZ	+	-	-	+	-	-		+						I	CAD,HT,DM	3.5	>3	
11	55/M	Old Lt Pre Rt	+	-	-	Partial / gen	-	-	+	+	CBZ	-	-	+	-	-	-	+	-	-		-	-	-	I	HT, DM	9	>3	
12	32/M	Lt	+	-	-	GTCS	-	-	+	+	CBZ	-	-	+	-	-	-		+	-		-	-		II		4.5	2	
13	75/M	Lt	+	-	-	GTCS	-	-	+	+	CBZ	+	-	-	-	+	-								III	HT, CAD	6	>3	
14	64/F	Rt	+	-	-	Partial / gen	-	-	+	-	PHT		+	-	-	-	+	+							III	DOM,DM	6	>3	
15	52/M	Lt	-	+	-	GTCS	-	+	-	-	PHT	+	-	-	-	-	-		+	-		-	+	L	I	HT	8.5		
16	63/M	Lt	+	-	-	Partial / gen	-	+	-		CBZ	+	-	-	-	-	+	+		-		-	-		IV	CAR,DM,HT	8	>3	
17	56/M	Rt	-	+	-	Partial	-	-	+	+	CBZ	+	-	-	-	-	-			-		-	+		III	DM,HT	3	>3	
18	62/M	Lt	-	-	+	GTCS	+	-	-	-	PHT	+	-	-	+	-	-			-		-	-	S	IV	RHD	1.5	>3	
19	75/M	Rt	+	-	-	Partial	+	-	-	-	CBZ	-	-	+	-	-	+		+	-		-	-		II	HT	9.5	2	
20	47/M	Lt	+	-	-	Partial	-	-	+	-	CBZ	+	-	-	+	-	+	+		-		-	-		III	CAD,HT	2.5	>3	

Sl.No	AGE / SEX	CVA (Side)	TYPE OF STROKE			Seizure Semiology	ONSET OF SEIZURE			Recurr	Choice of AED	ANATOMY			INFARCT			SIZE OF INFARCT		INTRA CEREBRAL HAEMORRHAGE				VOLUME OF ICH	EEG	Comorbid condition	CNS Score	Charlson Index	Death
			Thromb	Hgic	Emb		IMM	Early	Late			Cortical	Subcortical	Cortical + Subcortical	Frontal	Parietal	Occipital	Small	Large	Deep ICH + IVH	Deep ICH +Lobar	Deep ICH + IVH + Lobar	Lobar	Small / Large					
21	65/M	Rt	+	-	-	GTCS	+	-	-	-	CBZ	+	-	-	+	-	-		+	-		-	-		III	HT	3	1	
22	55/M	Lt	+	-	-	Partial / gen	-	-	+	+	PHT	+	-	-		-	+		+	-		-	-			DCM,DM,HT	9	>3	Early
23	35/M	Rt	+	-	-	Partial / gen	-	-	+	+	CBZ	+	-	-	-	-		+		+		-	-		I		6.5	>3	
24	60/M	Rt	-	+	-	GTCS	-	-	+	+	PHT	+	-	-			+			-		+		L		DM,HT,CKD	8.5	>3	Early
25	67/F	Rt	+	-	-	GTCS	+	-	-	-	CBZ	+	-	-	-	+	-		+	-		-	-		III		2.5	0	
26	35/M	Lt	+	-	-	Partial	-	-		-	CBZ	+	-	-		-	-	+		-		-	+		IV	HT,CAD	3	>3	
27	52/F	Rt	+	-	-	GTCS	-	-	+	+	CBZ	+	-	+	-		-		+	-		-	-			CAD,HT,RI,DM	8	>3	Late
28	32/M	Lt	-	+	-	Partial / gen	+	-	-	+	CBZ	+	-	-	-	-	+			-		-		L	III	HT	6.5	2	
29	45/M	Lt	+	-	-	Partial	-	-	+	+	PHT	-	+		-	+	-		+	-		-	-		IV	HT,DM,CKD	1.5	>3	
30	56/M	Rt	-	-	+	Partial / gen	+	-	-	-	PHT	+	-	-		-	-			-		-	+		III	HT,DM,CAD	2	>3	
31	60/M	Rt	+	-	-	Status	-	-	+		PHT	-	+	-					+			+			II		10	1	
32	58/M	Rt	+	-	-	GTCS	+	-	-	-	CBZ	-	+	-		+		+							III	HT,DM	2.5	>3	
33	56/M	Lt	-	+	-	GTCS	-	+	-	-	CBZ	-	+	-						-		-	-	L	I	CKD,HT,DM	3.5	>3	
34	75/M	Rt	+	-	-	Partial / gen	+	-	+	+	CBZ		+	-		+	-		+	-		-	-		V	CAD,HT	1.5	>3	
35	60/F	Lt	+	-	-	Partial / gen	+	-	-		CBZ	-		+		+	-		+	-		-	-		II	DM,HT	10.5	>3	
36	31/F	Lt	+	-	-	Partial	+	-	-		CBZ		+	-	-	+			+	-		-	-		III	CKD,HT	3	>3	
37	32/F	Lt	+	-	-	Partial	+	-	-	+	CBZ	+	-	-	-		-			+		-		S	IV		6.5		
38	67/M	Rt	-	+	-	GTCS	+	-	-		CBZ	+	-	-	-	-	-	+		-		-			III	RHD	6	>3	
39	50/F	Rt	-	-	+	Partial / gen	+	-	-	+	PHT	-	-	+	-	-				-		-	+	S		HT	8	>3	
40	62/F	Lt	-	+	-	GTCS	+	-	-		PHT	+	-	-	-	-	-	+		-			-	L	III	IV	4.5	>3	

Sl.No	AGE / SEX	CVA (Side)	TYPE OF STROKE			Seizure Semiology	ONSET OF SEIZURE			Recurr	Choice of AED	ANATOMY			INFARCT			SIZE OF INFARCT		INTRA CEREBRAL HAEMORRHAGE				VOLUME OF ICH	EEG	Comorbid condition	CNS Score	Charlson Index	Death
			Thromb	Hgic	Emb		IMM	Early	Late			Cortical	Subcortical	Cortical + Subcortical	Frontal	Parietal	Occipital	Small	Large	Deep ICH + IVH	Deep ICH +Lobar	Deep ICH + IVH + Lobar	Lobar	Small / Large					
41	70/m	Rt	-	+	-	Partial	+	-	-	+	CBZ		+	-	-	-	-			-		-	+		I	HT	9.5	2	
42	55/M	Rt	+	-	-	GTCS	+	-	-	+	CBZ	+	-	-	+	-	-			-		-	-			CAD,AF,HT,DM	3	>3	Early
43	60/M	Lt	+	-	-	GTCS	-	-	+		PHT	+	-	-	-	+	-			-		-	-		IV	IV	9	1	
44	62/M	Rt	+	-	-	GTCS	+	-	-	+	CBZ	-	+	+	-	-	-			-		-	-		III	HT	2.5	>3	
45	47/F	Old Rt Pre Lt	+	-	-	Partial	-	-			CBZ	+	-	-	-	+	-			-		-	-		IV	HT,CAD,DM,CKD	7	>3	
46	40/M	Lt	-	-	+	GTCS	+	-	-		CBZ	+	-	-	+	-	-			-		-	-			RHD	2.5	>3	
47	32/M	Lt	+	-	-	Partial	-	-	+	+	PHT	+	-	-	-	-	+			-		-	-		III	DM,CAD	3	>3	
48	56/M	Rt	-	+	-	Partial	+	-	-		CBZ	-	+	-	-	-	-			-		-	-		III	HT	9	2	
49	56/M	Lt	+	-	-	CPS	-	-	+	+	CBZ	+	-	-	-	-	+			-		-	-			DM,CAD	4.5	>3	
50	55/M	Lt	-	-	+	Partial / gen	-	-	+	+	CBZ	+	-	-	-	+	-			-		-	-		I	RHD	6	>3	
51	56/F	Rt	-	+	-	GTCS	+	-	-		CBZ	+	-	-	-	-	-			-		-	+		III		8	0	
52	39/F	Aphasia	-	-	+	GTCS	+	-	-		PHT	-	-	+											IV	RHD	3	>3	
53	51/M	Rt	+	-	-	Partial	-	-	+		CBZ	+	-	-	-	-	+			-		-	-		III	CKD,DM,HT	7.5	>3	
54	41/F	Lt	+	-	-	Partial	+	-	-	-	CBZ	+	-	-	-	+	-			-		-	-		IV		2.5	2	
55	62/M	Lt	-	+	-	Partial	-	-	+		CBZ	+	-	-	-	-	-			-		-	+			DM,HT,CKD	10	>3	
56	29/M	Rt	-	+	-	Partial / gen	-	-	+		CBZ	-	-	+	-	-	-			-		+	-		II	DM,CAD	4.5	>3	
57	44/M	Rt	+	-	-	GTCS	+	-	-		CBZ	+	-	-	-	+	-			-		-	-		IV	HT,CAD	4	>3	
58	72/F	Lt	-	-	+	Partial	+	-	-	+	PHT	-	-	+											III	Hyper Thyroid / AF,HT	11	>3	
59	67/M	Lt	-	-	+	GTCS	+	-	-	+	PHT	+	-	-	-	+	-			-		-	-		IV	RHD	8	>3	
60	62/M	Rt	+	-	-	GTCS	+	-	-		CBZ	+	-	-	-	+	-			-		-	-		I		3.5	1	

Sl. No	AGE / SEX	CVA (Side)	TYPE OF STROKE			Seizure Semiology	ONSET OF SEIZURE			Recurr	Choice of AED	ANATOMY			INFARCT			SIZE OF INFARCT		INTRA CEREBRAL HAEMORRHAGE				VOLUME OF ICH	EEG	Comorbid condition	CNS Score	Charlson Index	Death
			Thromb	Hgic	Emb		IMM	Early	Late			Cortical	Subcortical	Cortical + Subcortical	Frontal	Parietal	Occipital	Small	Large	Deep ICH + IVH	Deep ICH +Lobar	Deep ICH + IVH + Lobar	Lobar	Small / Large					
61	78/M	Lt	+	-	-	Partial	-	-	+		CBZ	+	-	-	-	+	-			-		-	-			HT, DM, Hyponatremia ,CAD,CKD	9	>3	
62	68/M	Lt	-	+	-	GTCS	+	-	-		CBZ	-	+	-						-		-	-				8	2	
63	72/M	Lt	+	-	-	Partial	-	+	-		CBZ	-	-	+						-		-	-			DM,CRD,HT,CAD	4	>3	
64	56/M	Rt	-	+	-	Partial	-	-	+		CBZ	+	-	-	-	-	-			-		-	+		III	DCM,DM	4	>3	
65	60/M	Rt	+	-	-	GTCS	-	-	+	+	CBZ	-	-	+												HT	9		Early
66	70/M	Lt	-	+	-	Partial / gen	-	+	-		CBZ	+	-	-	-	-	-			-		-	+		I	DM,HT,CKD	3	>3	
67	46/F	Rt	-	+	-	Partial	-	+	-		CBZ	-	+	-	-	-	-			-		-	-		III		7	2	
68	28/F	Rt	-	-	+	Partial	+	-	-		CBZ	+	-	-	-	+	-			-		-	-		III	RHD	8	>3	
69	60/M	Lt	+	-	-	Partial	-	-	+		CBZ	+	-	-	+	-	-			-		-	-		II	DM,HT,CKD	4.5	>3	Late
70	45/M	Rt	+	-	-	Partial	-	-	+	+	CBZ	+	-	-	-	+	-			-		-	-				3.5	>3	
71	50/M	Lt	+	-	-	Partial	+	-	-		CBZ	+	-	-	-	+	-			-		-	-		III	HT,DM	9.5	>3	
72	40/M	Rt	-	+	-	GTCS	+	-	-		CBZ	-	+	-	-	-	-			-		-	-			HT,CKD	6	>3	
73	62/M	Rt	+	-	-	Partial / gen	+	-	-		CBZ	-	+	-													11	2	
74	50/F	Lt	-	+	-	Partial / gen	+	-	-		CBZ	-	+	-	-	-	-			-		-	-		III	CKD,HT,DM	7	>3	
75	52/F	Lt	-	-	+	Partial / gen	-	-	+	+	CBZ	+	-	-	+	-	-			-		-	-			RHD	8	>3	
76	70/M	Rt	-	+	-	Partial / gen	-	-	+	+	CBZ	+	-	-	-	-	-			-		-	+		IV		5	1	
77	46/M	Rt	-	+	-	GTCS	+	-	-		CBZ	-	-	+	-	-	-			-		+	-		III	HT	6	>3	
78	68/F	Rt	+	-	-	Partial	+	-	-		CBZ	+	-	-	-	+	-			-		-	-		I		7.5		
79	56/F	Lt	+	-	-	Partial / gen	+	-	-		CBZ	-	+	-												HT,DM	5	>3	
80	48/M	Rt	-	+	-	Partial / gen	-	+	-		CBZ	-	+	-	-	-	-			-		-	-		III	HT,CAD,DM,CKD	4	>3	
81	40/M	Lt	+	-	-	Partial / gen	+	-	-	+	CBZ	+	-	-	-	+	-			-		-	-				10	1	

Sl. No	AGE / SEX	CVA (Side)	TYPE OF STROKE			Seizure Semiology	ONSET OF SEIZURE			Recurr	Choice of AED	ANATOMY			INFARCT			SIZE OF INFARCT		INTRA CEREBRAL HAEMORRHAGE				VOLUME OF ICH	EEG	Comorbid condition	CNS Score	Charlson Index	Death
			Thromb	Hgic	Emb		IMM	Early	Late			Cortical	Subcortical	Cortical + Subcortical	Frontal	Parietal	Occipital	Small	Large	Deep ICH + IVH	Deep ICH +Lobar	Deep ICH + IVH + Lobar	Lobar	Small / Large					
82	68/M	Rt	+	-	-	Partial / gen	+	-	-		CBZ	+	-	-	-	+	-			-		-	-		III		6.5	>3	
83	60/F	Lt	-	+	-	GTCS	+	-	-		CBZ	+	-	-	-	-	-			-		-	+			CAD,HT,DM	2	>3	
84	80/M	Rt	-	+	-	GTCS	-	-	+		CBZ	+	-	-	-	-	-			-		-	+		I		8.5	>3	
85	36/M	Lt	-	-	+	Partial	-	-	+	+	CBZ	+	-	-	+	-	-			-		-	-		III	RHD	3		
86	58/M	Lt	+	-	-	GTCS	+	-	-		CBZ	+	-	-	-	+	-			-		-	-		III	CAD,DM	4.5	>3	
87	62/M	Lt	+	-	-	Partial / gen	-	+	-	+	CBZ	-	-	+												HT,CKD	5	>3	
88	68/M	Rt	+	-	-	Partial / gen	+	-	-		CBZ	+	-	-	-	+	-			-		-	-		IV		6.5	2	
89	68/M	Rt	-	+	-	Partial	-	-	+		CBZ	+	-	-	-	-	-			-		-	+		III	CKD,HT,DM	11	>3	
90	71/M	Rt	+	-	-	Partial	+	-	-	+	CBZ	+	-	-	-	+	-			-		-	-		I		4	2	
91	48/M	Rt	-	+	-	Partial / gen	-	-	+	+	CBZ	+	-	-	-	-	-			-		-	+		IV	HT	7.5	0	
92	69/M	Rt	+	-	-	Partial	-	+	-		CBZ	-	-	+												HT,DM	6.5	>3	
93	46/M	Rt	+	-	-	Partial	+	-	-		CBZ	+	-	-	+	-	-			-		-	-		III	DCM	9	>3	
94	80/M	Lt	+	-	-	GTCS	-	-	+	+	CBZ	+	-	-	-	+	-			-		-	-			HT,DM	4	>3	Early
95	72/M	Rt	+	-	-	Partial / gen	-	-	+	+	CBZ	+	-	-	-	+	-			-		-	-		V	HT,CAD,DM	9	>3	
96	54/M	Rt	-	+	-	GTCS	-	+	-		CBZ	+	-	-	-	-	-			-		-	+	S	III	CKD,HT,DM	4	>3	
97	68/M	Lt	-	+	-	Partial	-	+	-		CBZ	-	+	-	-	-	-			-		-	-	S	I	HT,CAD	3.5	>3	
98	63/M	Rt	-	+	-	Partial	-	-	+	+	CBZ	-	-	+	-	-	-			-		+	-	S	II	CKD,DCM,DM	7.5		
99	56/F	Rt	+	-	-	GTCS	+	-	-		CBZ	+	-	-	-	+	-			-		-	-		I	RHD		>3	
100	48/F	Rt	-	-	+	Partial / gen	-	-	+	+	CBZ	+	-	-	+	-	-			-		-	-			RHD		>3	